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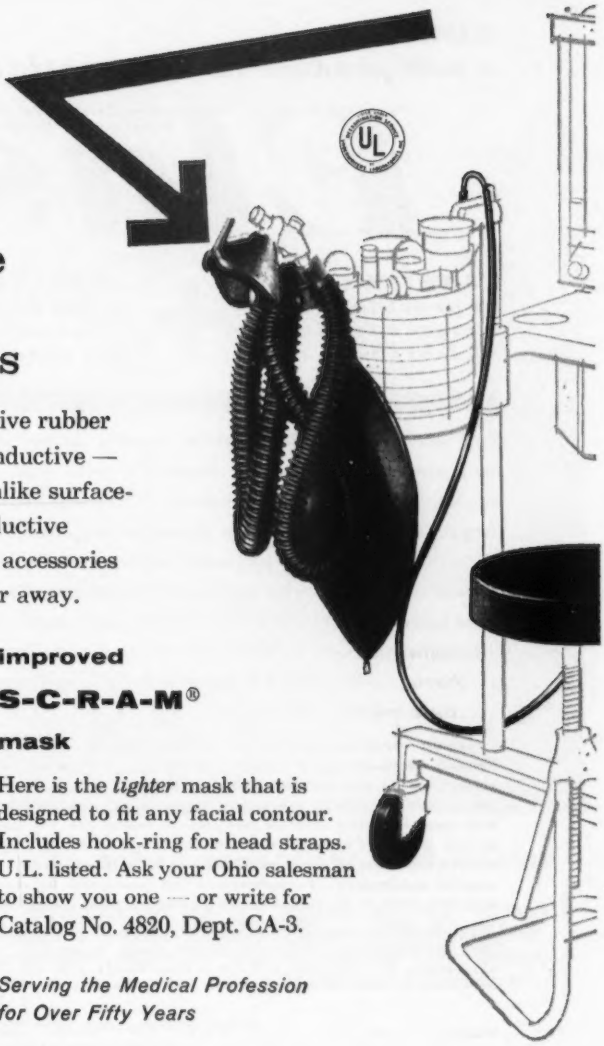

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CLINICAL MEDICINE AND SURGERY IN ANAESTHETIC PRACTICE*

JOHN GILLIES, C.V.O., M.C., M.B., F.R.C.P.E., F.R.C.S.E., F.F.A.R.C.S., HON. F.F.A.R.A.C.S., HON. F.F.A.R.C.S.I.

THE CIRCUMSTANCES of the foundation of the Shields Lecture have gratified anaesthetists far beyond Toronto and Canada and particularly those among us who have been privileged to know the man whose work will now be commemorated in perpetuity. Such signal appraisal rarely comes in a man's lifetime, but surely, to one so self-effacing and so worthy of the homage of his colleagues and of generations of patients as Harry Shields, the gesture was both timely and appropriate. It has crowned a dignified career of outstanding merit which has contributed notably to the recognition of anaesthetic practice as a distinctive speciality, not only within the profession but also in the minds of a grateful laity.

It was a considerable act of faith on the part of the leaders in the speciality of anaesthetics in Toronto and of the Dean and Faculty of its world-famed medical school to invite me to deliver the second Shields Lecture. As the first non-Canadian so honoured, I became at once deeply aware of the responsibility that would devolve upon me and I must confess frankly that sentimentality overruled cold reasoning in prompting my decision to accept. That is perhaps excusable in one who, nearing the end of his professional life, cannot proffer the scientific pabulum for which present-day anaesthetists appear to have a considerable craving, but thinks, possibly naïvely, that he may still have something to pass on.

After five days of listening to or participating in the academic discussions at the Second World Congress of Anaesthesiologists, most of you may be more than ready to descend with me from the Olympian heights and tolerate or even find relaxation in mental anti-climax. On this occasion, therefore, I trust there is no need to apologize for speaking on workaday matters which must forever be of importance to all who practise the most critical and most responsible form of applied pharmacology in the whole field of medicine.

Within the limits purposely implied in the title of this paper, I feel committed mainly to a discussion of certain surgical and medical conditions concerning which anaesthetists should have more than a cursory knowledge if they aspire to completeness in their work and to the respect of their colleagues in other branches of medicine. Concurrently, consideration will be given to the professional relationships involved in the broadening of the range of anaesthetic practice.

EVOLUTION OF MODERN PRINCIPLES

Thirty or more years ago little was expected of the anaesthetist beyond doing his best to keep the patient immobile, to provide a modicum of relaxation—a

*Second Shields Lecture, delivered at the University of Toronto, September 9, 1960.

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little more was tacitly but doubtless gratefully acknowledged—and to get the patient off the operating table alive. Effecting such a compromise was not easy, particularly in respect to the increasingly ambitious upper abdominal surgery of that time when, apart from nitrous oxide which was worse than useless, the general anaesthetics available were powerful depressants of vital functions if given in the toxic concentrations necessary to produce reasonable operating conditions. The price paid for the latter was an almost prohibitory incidence of respiratory and circulatory complications and no kudos for the anaesthetist. It is no wonder, therefore, that in the period between the first and second world wars many surgeons, particularly in the clinics of continental Europe, were loath to exchange non-toxic nerve blocks for general anaesthesia when the former were serving so well on account of their safety and anti-shock qualities, and apparently facilitating rather than hindering surgical progress. But for the timely introduction of muscle-relaxants I believe that the use of local and regional analgesia would have become universal. Anaesthetists, however, having meantime made themselves indispensable through their skill in tracheal and bronchial intubation and controlled respiration would not have lost their jobs because such valuable procedures would continue to be necessary, even if nerve blocks came into fashion again. As one who has seen many advances in surgical and anaesthetic practice, I venture at this point to prophesy that good as present-day anaesthetic methods are for patient and surgeon, and, challenging and titillating as they may be for many anaesthetists, the trend in the not too distant future will be towards an increasing use of local and regional nerve blocks accompanied by controlled depression of consciousness and efficient mechanical ventilation of the lungs. I am all for automation and no intoxication.

More than a generation ago Crile demonstrated the merits of a non-toxic combination of local analgesia and unconsciousness. His principle of "Anoci-association" can be applied with even greater effect to-day because of the better equipment available for maintaining a uniform light level of unconsciousness and satisfactory pulmonary ventilation. In my experience patients undergoing extensive and stress-inducing surgery are best served by some form of nerve block plus light anaesthesia as exemplified by the operation record (Fig. 1) in the case of a girl of 18 years in whom a hindquarter amputation was performed for sarcoma.[1] Protection against stress-producing trauma at the site of operation was provided by spinal blockade. The accompanying complete sympathetic paralysis prevented circulatory reflex activity, ensured stability of the blood pressure at the intended hypotensive level, and obviated loss of blood. No transfusion was given because none was necessary and, as may be noted, no time was wasted by the surgeon, Sir Gordon Gordon-Taylor, whose tragic death in London is deplored by the entire medical world.

The period of technical development, adorned by virtuosi such as Ralph Waters and Ivan Magill, brought initially hesitant but significant recognition to the speciality of anaesthetics, at first strictly limited to individuals but later to many who followed the leaders and similarly impressed their surgical colleagues with their dexterity. It was natural that surgical craftsmen should appreciate craftmanship in others, but physicians who only got occasional glimpses of us at

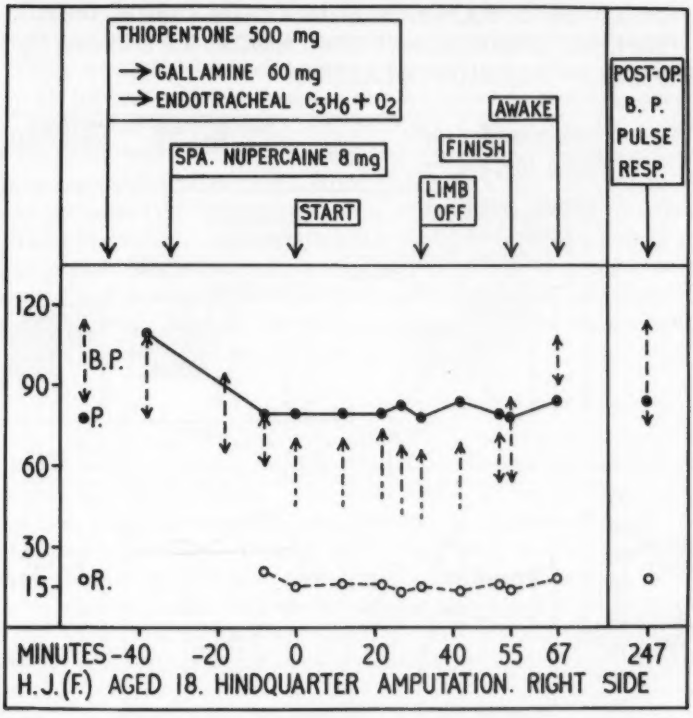

FIGURE 1

work perhaps thought that we were merely being crafty. With commendable liberality of mind, doubtless in token of benefits received and possibly in anticipation of more to come, many of our surgical colleagues of that time entrusted us with an increasing responsibility in the care of their patients and thus hastened the era of "physiological trespass."[2] This progressive three-stage concept comprising curarization, induced hypotension, and hypothermia introduced successively over a few years was found difficult to swallow, particularly its second component, by many surgeons and some anaesthetists who sided with them. As so often happens, however, having once tasted, their appetites became excessive and for a time the usual trail of teething troubles ensued, but on a scale almost big enough and serious enough to halt progress. It is extraordinary how enthusiasm can obliterate common sense!

Now, at long last, despite setbacks sometimes generated by anaesthetists themselves, the practice of anaesthetics, at least in its technological aspects, was able to provide conditions that would enable surgeons to explore any region of the body with reasonable safety and have adequate time to carry out remedial and reconstructive procedures particularly on the heart and great vessels. And it all turned out to be so simple in the end—dangerously simple indeed, so that even

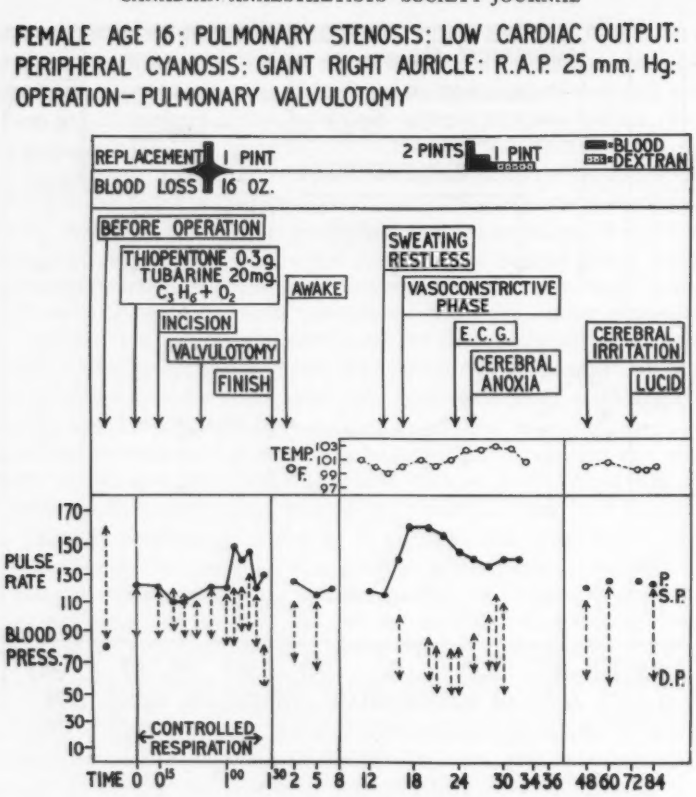

FIGURE 2

the novice produced as acceptable results as his seniors. Could this be enough? Surely not! The complexity of certain operations on the cardiovascular system and on the brain required from anaesthetists, as indeed it did from surgeons, something more than skilled technology. If they had not found it particularly necessary before, anaesthetists now had to acquire a physician's knowledge of circulatory, respiratory, and endocrinal pathology. But further than that they had to learn *de novo* the physiological readjustments that take place during and after surgical correction of congenital and acquired diseases and after the extirpation of organs on which homeostasis and life itself depends. The need for such study and preparedness is illustrated in the anaesthetic record (Fig. 2) of a girl of 16 years undergoing pulmonary valvotomy for pulmonary stenosis associated with a giant right auricle in which the preoperative pressure was 25 mm. Hg. This compensatory increase had enabled the heart to maintain an adequate basic output for some years. Blood lost during operation was fully replaced at the time but some hours later the patient became hypotensive although there was no evidence of further blood loss. An electrocardiogram showed sinus tachycardia,

not ventricular tachycardia as at first diagnosed. The blood pressure continued to fall probably as a result of the decrease in right auricular pressure and in cardiac output which followed relief of the stenosis and a sudden, but significant, alteration in haemodynamics. Progressive circulatory failure was checked by infusion of blood and dextran and the hypotension became reversed, presumably because the blood volume, the intra-auricular pressure, and the cardiac output had all been augmented.[1] Here was a situation in which over-transfusion could be justified as a means of tiding a patient over a period of cardiovascular adjustment. As anaesthetist-physicians we must, therefore, be alert to the hazardous unstable circulatory states which may follow surgical correction of cardiac anomalies, particularly those associated with pulmonary hypertension, and keep in mind that adaptation of the heart to altered circumstances may be relatively slow and that circulatory collapse is liable to develop meantime.

Emergency Surgery

It is in the field of emergency surgery that anaesthetists find most scope for a sound knowledge of pathology. The importance of emergency cases being examined and, wherever possible, anaesthetized by experienced staff members cannot be over-emphasized. Through familiarity with surgical conditions and the varying impact of operative procedures, an understanding of intercurrent pulmonary, cardiovascular, and endocrinal diseases in relation to anaesthesia and, by no means least, acquaintance with the methods and capabilities of their operating colleagues, anaesthetists should be better equipped than most physicians to assess the fitness of patients for operation and the attendant over-all risk, and to advise on appropriate preparation. In brief, without aspiring to be fully expert in internal medicine, or usurping the place of the physician, anaesthetists ought to maintain a studious interest in medical conditions, particularly those commonly occurring intercurrently in surgical cases.

Urgency

Of the various general aspects of emergency surgical treatment, I propose to deal only with the question of urgency since it is in this matter that the views of surgeons and anaesthetists may clash, although, with a sensible understanding of each other's problems, there should rarely be any disagreement. Urgency varies with the extent and the rate at which physiological function and homeostasis are deranged by the surgical lesion or by intercurrent disease. Thus, there is extreme urgency when pulmonary ventilation is failing rapidly from various causes, particularly thoracic trauma and raised intracranial tension. Urgency is immediate if, on account of continuing haemorrhage, circulatory collapse is imminent, or, in a somewhat different clinical field, when a foetus is in distress and hysterotomy or forceps delivery becomes suddenly necessary.

Common conditions such as gastrointestinal obstruction, visceral inflammation and perforation, and acute urinary retention are relatively less urgent and time is usually available for more adequate preoperative preparation of patients in this category. Time, however, must not be wasted and the anaesthetist should

always consider carefully his wider clinical responsibility when advising or causing any delay and have sound reasons for whatever course of action he may suggest or pursue.

Trauma

The treatment of traumatic conditions forms the major part of urgent or semi-urgent surgery at the present time. Rapid transportation on overcrowded highways and airways contributes increasingly to this sinister state of affairs. Even more so than in the acute surgical diseases, the seriousness of trauma is related to the degree of discontinuity of vital functions, particularly circulation and respiration. In the urgent circumstances which frequently prevail, all the highly developed skills of the anaesthetist and his knowledge of surgery and medicine are put to the test.

Crush Injury of Chest

Apart from vascular disruption leading quickly to death, the most serious injuries are multiple fractures of ribs accompanied by respiratory paradox. This condition may be rapidly fatal even after the patient is supposedly under control. It is simple enough to recognize and deal with paradox associated with an open wound of the thorax but, if the opening is valvular in character, or, more insidiously, when an unsuspected traumatic bronchopleural fistula is present with the thoracic wall remaining intact, an immediate appreciation of the general situation is essential to obviate disaster. The resultant fulminating tension pneumothorax may be unwittingly aggravated by controlled respiration. Diagnosis of such a grave development in an anaesthetized patient depends on recognition of increasing difficulty in inflating the lungs, uncorrectable cyanosis, rising blood pressure and pulse rate, and displacement of the apex beat, following one another in dramatic crescendo.

Dr. Griffiths, a member of my department, has devoted much time during recent years to investigating the apparently unnecessarily high mortality and morbidity associated with multiple injuries including crushed chest and his detailed study of the problems associated with such accidents will be published soon.[3] In our departmental discussion on the subject we are agreed that the essential preliminary is an immediate assessment of priorities in respect to the order of treatment of the several injuries and we consider that, in the first instance, this should be entrusted to a general surgeon along with an anaesthetist. All too often, attention to the commonest and most grave complication, namely, respiratory insufficiency, is left until too late while measures less urgent in respect to the saving of life are being instituted. Whenever possible the treatment of a severe chest injury should begin at the scene of the accident because traumatic pulmonary and haemodynamic dysfunction kills quickly.

Figure 3 illustrates a case of multiple injuries in which persistent treatment of circulatory depression was given priority over treatment of pulmonary dysfunction and was unsuccessful until effective ventilation was established.[3] Three phases in mangement may be noted.

(1) In spite of "wet" lungs, deviation of the trachea, and laboured respiration

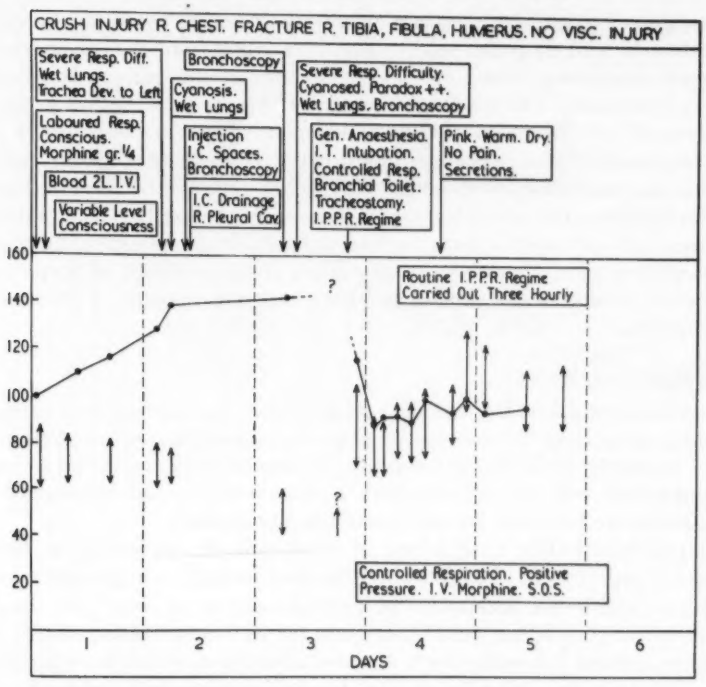

FIGURE 3 (By courtesy of Editor, Jour. Roy. Coll. Surg. Edin.)

doubtless aggravated by morphia, treatment in the first phase was concentrated on improving the blood pressure by direct means. This was unsuccessful and the patient's condition deteriorated as indicated by a continuous rise in pulse rate and no improvement in blood pressure.

(2) Measures to deal with pulmonary dysfunction were instituted, namely, pleural drainage and bronchoscopic aspiration, but by this time these were not sufficient to arrest a dangerous decline towards circulatory failure.

(3) An active régime of intermittent positive pressure respiration through a tracheostomy to restore and maintain full ventilation of the lungs was established just in time to save the patient's life.

In his review,[3] Griffiths has shown that in cases of multiple injuries to thorax, head, limbs, and abdominal viscera the commonest cause of death is respiratory insufficiency and that frequently this is avoidable. The immediate correction of respiratory dysfunction, when acute or when sub-acute and insidious, should, therefore, have the top priority, sharing this if need be with the arrest of continuing haemorrhage. Treatment of fractures, even mechanical fixation of the ribs, which is not commonly necessary, can wait and quite often circulatory depression and unconsciousness along with other neurological signs caused by hypoxia and carbon dioxide retention will disappear once respiratory function is normal.

If respiratory insufficiency is not effectively treated, hypoxia, retention of carbon dioxide, and dyspnoea lead to a shift of blood from the periphery to the pulmonary circulation which may be augmented if the patient has been too zealously transfused. The resulting pulmonary hypertension and congestion will be aggravated by absorption collapse of segments of the lungs and grave pathological changes will ensue, namely, raised capillary pressure, oedema, turgidity, and reduced compliance of the lungs. This will be followed by increased respiratory effort and negative endopleural pressure, more transudation from pulmonary capillaries, and still more oedema to continue the vicious spiral leading to death. In primarily surgical conditions of this kind, there is plenty of scope for the anaesthetist to exercise his skill as an interpreter and assessor of physical signs and symptoms.

Haemorrhage

Another common and often critical problem for anaesthetists and surgeons is impending circulatory failure due to a marked reduction in circulating blood volume, especially when this is the result of massive bleeding. Anaesthetists are much concerned with the management of this condition and particularly with the decision as to how soon surgery should be undertaken.

A clinical record (Fig. 4) of a case of uncontrollable haemorrhage illustrates the factor of urgency in a situation which brooked no delay whatsoever in surgical intervention and in the acceptance of a calculated risk in respect to the anaesthetic procedure.[1] A young man, exsanguinated as a result of a gunshot wound of the liver, arrived in hospital forty minutes after the accident. He was conscious but showed signs of cerebral hypoxia; his pulses were impalpable, his blood pressure was unrecordable, and he was gasping. The patient was taken directly to the operating theatre where a rapid blood transfusion was started and, simultaneously, anaesthesia was induced with cyclopropane and oxygen. Fifteen minutes after admission laparotomy was being performed and the disrupted liver sutured. Not until completion of the operation did the blood pressure become recordable. This remained at about 60 mm. Hg for over one hour after which blood replacement appeared to be effective. The patient became conscious and lucid and ultimately his blood pressure attained a level of 120–70 mm. Hg with a fall in pulse rate from 150 to 110 per minute. Ominously, however, the pulse quickened and the blood pressure fell as a result of inevitable further bleeding. The circulation was improved again by increasing the speed of transfusion but the improvement was more apparent than real; the pulse rate remained fast and the patient died five hours later. Such an outcome had seemed likely when the grave nature of the visceral and vascular damage was fully revealed by laparotomy. Immediate operation to stop severe continuous bleeding, although only transiently effective in this case because it could not be otherwise, gave the patient his one remote chance of survival.

Immediate Diagnosis

In contrast to elective surgery where preoperative investigations can be as complete as need be, truly urgent surgery does not permit time for the use of

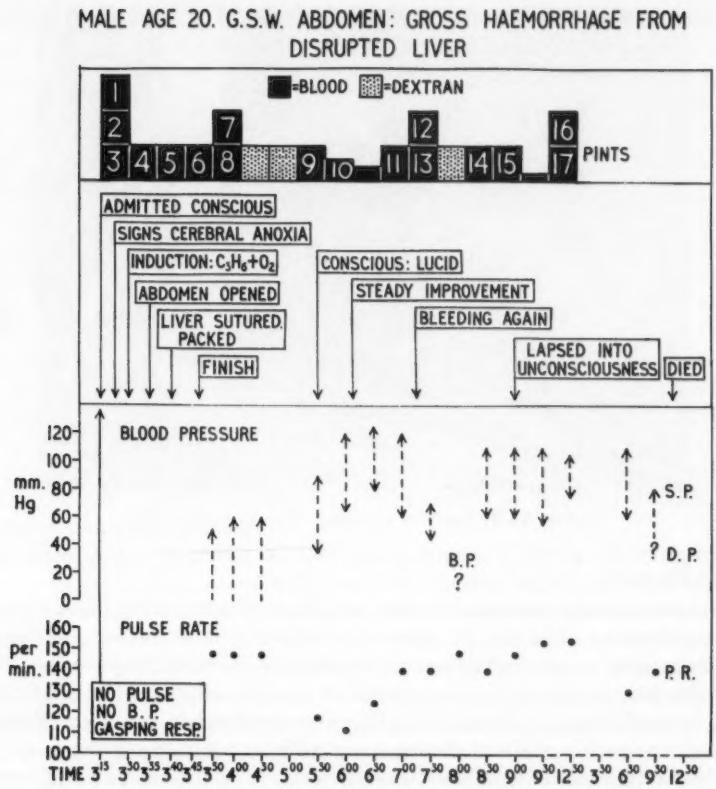

FIGURE 4

modern aids, diagnostic and otherwise, and in such circumstances clinical acumen is all important and anaesthetists bear a heavy responsibility in respect to the survival of ill, unprepared patients. In immediate clinical diagnoses they must be acutely aware of the possibilities of mistaking coronary thrombosis for perforated peptic ulcer, biliary colic, or acute pancreatitis, or adrenal apoplexy for internal haemorrhage. Their knowledge of acute and chronic pathological conditions should be such as will enable them to make an intelligent and tactful contribution in discussing with surgical colleagues problems in differential diagnosis, resuscitation, and management, without straining personal relations. The anaesthetist's outlook must be as wide-angled as possible and not focussed solely on anaesthetic matters. With the non-toxic drugs and procedures available nowadays, he can make the patient much safer for operation than could his predecessors and so raise the operability rate in dangerously ill patients. Where competent he may offer help in certain aspects of the care of the patient and often this may be appropriate when the surgical partner is a junior with less general clinical experience than himself.

FIGURE 5 (By courtesy of Editor, Brit. Jour. Anaesth.)

Surgical Diseases

Some other acute conditions in which problems arise requiring frank discussion and co-ordination of action by surgeon and anaesthetist merit consideration:

(1) Relatively minor imflammatory conditions are sometimes serious anaesthetic risks but are not always recognized as such. Examples of this are Ludwig's angina, retropharyngeal abscess, and infective swellings in the neck. If there is much oedema within the oral pharynx and difficulty in opening the mouth, the anaesthetist must not delude himself or yield to the suggestion of a well-meaning surgeon that a muscle relaxant might solve the problem. This way, and particularly if an intravenous barbiturate is given about the same time, there will almost certainly be a point of no return for the patient. In an age of easy anaesthesia it must be emphasized that one must never get a patient into a state which is irreversible or can only be reversed with so much difficulty and lapse of time that life or well-being remains in jeopardy.

The patent airway is a hackneyed subject but here I should like to mention a particular cause of respiratory obstruction that may be rare but certainly is seldom diagnosed. I refer to narrowing of the glottis as a result of rheumatoid arthritis involving the arytenoids. For the microscopic section (Fig. 5) I am indebted to my colleagues D. L. Gardner and Frank Holmes who have been investigating this interesting pathological condition.[4] The characteristic histological features are lymphocytic infiltration of the cartilages and synovial membrane with oliteration of the joint space. Realizing the potential danger of this condition and of some more obvious ones such as trauma of the bones and soft tissues of the face accompanied by oedema, serious consideration should be given to tracheostomy as a justifiable life-preserving precaution before induction of anaesthesia.

Concerning tracheostomy in general, anaesthetists have so far been more zealous than their colleagues in advocating this measure not only in certain surgical cases but also for medical conditions involving respiratory insufficiency and the circulatory and cerebral depression associated with progressive sub-acute asphyxia. In this connection there is still a considerable field in medical units for exploiting ancillary skills of anaesthetic practice such as bronchoscopic aspiration and the use of mechanical respirators.

(2) A major surgical emergency seen relatively often in hospital practice although surprisingly rarely by individual general medical practitioners is perforated peptic ulcer. The typical clinical features of this condition are familiar to experienced anaesthetists but there are two problematical points of peculiar interest which merit discussion.

The first concerns the differential diagnosis of myocardial infarction which may be mistaken for perforated peptic ulcer, acute pancreatitis, dissecting aneurysm of the thoracic aorta, pneumonia, or pulmonary embolism. Occasionally a case of myocardial infarction is admitted to hospital as an abdominal emergency, probably as a perforated viscus, and in such circumstances the anaesthetist, as physician to the surgical unit, may, quite properly, be expected to indicate the alternative non-surgical diagnosis.

The misleading symptom is pain, or, perhaps more correctly, its interpretation. When it occurs in association with acute myocardial infarction, pain is sudden in onset, is located behind the sternum but sometimes in the epigastrium, and frequently radiates to the shoulders and arms, particularly on the left side. It may be accompanied by nausea and vomiting and this syndrome suggests an acute abdominal condition. The patient becomes hypotensive and pallid, dyspnoeic and somewhat cyanosed; muscular rigidity is absent or insignificant in degree.

In most but not all cases of perforated peptic ulcer a history of previous gastric trouble can be elicited. Pain starts suddenly in the epigastrium and is severe in character. It may occasionally be referred to the lowest part of the chest but never to the arms. Respiration is shallow and costal in type because of voluntary restriction to reduce pain; the dyspnoea is not so severe as in acute myocardial infarction. In cases of perforation hypotension, pallor and cyanosis appear later as classical manifestations of generalized peritonitis. Here also, radiographs, taken if possible with the patient sitting upright, will usually reveal the presence of gas under the diaphragm and sometimes areas of pulmonary collapse.

Finally, electrocardiography, although often inconclusive, at an early stage may clinch the diagnosis and should be carried out whenever there is any doubt since an unnecessary anaesthetic and laparotomy could be a grave additional hazard for a patient suffering from acute myocardial infarction.

The second feature is the high incidence of respiratory complications associated with perforated peptic ulcer. Until recently it has been generally accepted that surgical and anaesthetic factors, with perhaps some extra bias against the anaesthetic, share the etiological responsibility in this matter. Both surgeon and anaesthetist, however, can take comfort in the fact demonstrated by Le Roux[5] that neither of them is responsible since the pulmonary pathology can be an almost immediate consequence of the actual perforation and is often present

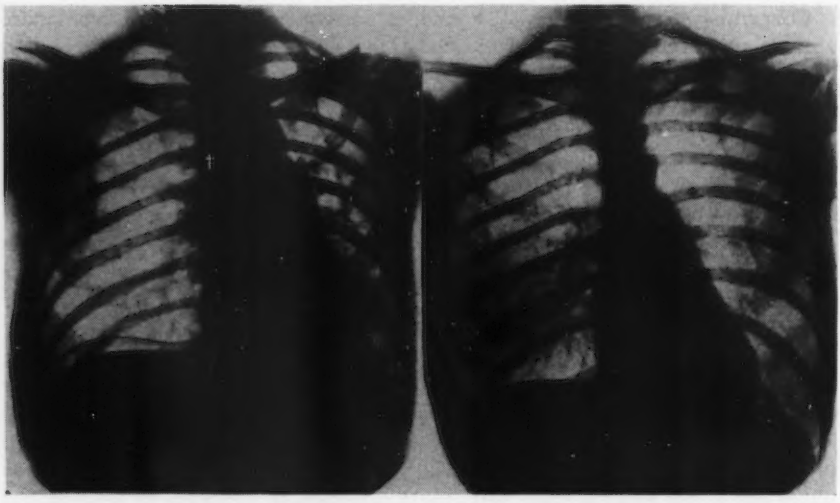

FIGURE 6 (By courtesy of Editor, Brit. Jour. Surgery)

before either of them has seen the patient. In illustration of this I quote two cases from a series of thirty described by Le Roux.

The first patient, aged 29, was admitted three hours after the onset of severe abdominal pain. The radiograph (shown on the left in Fig. 6) made on admission showed a pneumoperitoneum and patchy collapse of the left lung. On the second day after closure of a perforated ulcer and immediate postoperative bronchscopic aspiration the pulmonary opacities had cleared (Fig. 6, on the right).

The second patient, a 24-year-old man, was admitted four hours after perforation of a duodenal ulcer. Immediate radiography showed pneumoperitoneum and a shrunken, airless, lower lobe of the right lung (Fig. 7, left). Before and after closure of the perforation the bronchi were cleared of secretions. A radiograph on the day after operation showed normal aeration of the formerly collapsed lobe (Fig. 7, right).

In 9 of 16 patients suffering from perforated peptic ulcer, preoperative radiographs showed lobular collapse, and two, lobar collapse. From these Le Roux concluded that (*a*) pulmonary lesions are common soon after actual perforation of peptic ulcers, and (*b*) such lesions are a result of the perforation. The pain of the latter is much more severe than that which follows laparotomy and the consequent restriction of movement of the ribs and diaphragm predispose to pulmonary collapse. In addition, the irritant gastric juice provides a strong stimulus to the parietal peritoneum of the upper abdomen, the receptor field of reflex respiratory inhibition, and so causes a marked reduction in pulmonary ventilation. Furthermore, mechanism for clearing secretions come virtually to a standstill at the moment of perforation and the significance of this is greater in bronchitic patients and smokers. Le Roux concludes that (i) a postero-anterior radiograph should be taken preoperatively with the patient erect (this is more

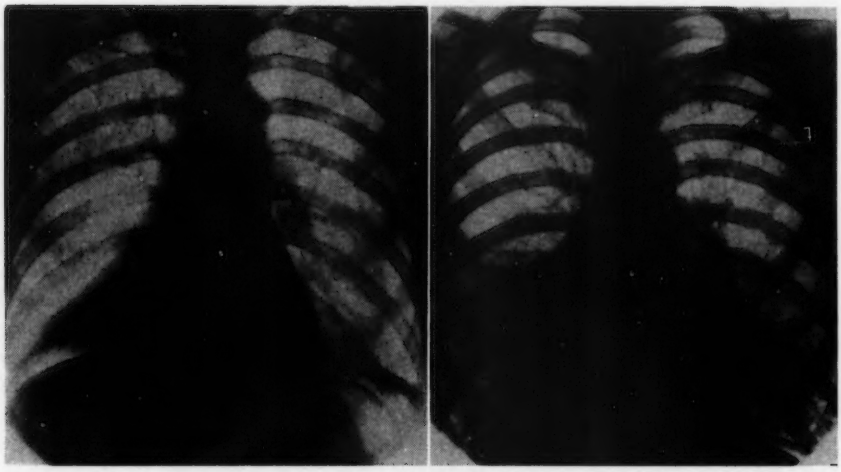

FIGURE 7 (By courtesy of Editor, Brit. Jour. Surgery)

helpful than physical examination when respiratory movements even in patients under analgesics are voluntarily inhibited), and (ii) aspiration of bronchial secretions after operation for perforated peptic ulcer and before if necessary is a sound and justifiable prophylactic measure.

In dealing with cases of perforation, therefore, anaesthetists must be prepared for the preoperative pulmonary complication of lobular or lobar collapse and undertake the initial responsibility for preventing its further development.

INCIDENTAL PROBLEMS

During surgical and obstetrical procedures under anaesthesia of any kind, complications of varying degrees of significance may develop inadvertently. Some are related to the operation and others to anaesthesia but occasionally, when a complex of circumstances is present, there may be no obvious line of demarcation. In whatever way it may originate, the situation is frequently a challenge to the vigilance, clinical awareness, and diagnostic capabilities of the operating team, not least the anaesthetist. The latter has his own iatrogenic problems to contend with, but it is not the purpose of this lecture to discuss these or the technology of present-day practice except to say that the sounder his scientific and clinical knowledge the easier it should be for the anaesthetist to assess his contribution, if any, to incidental complications. For his own sake, however, he must also be cognizant of other possible factors not related to the anaesthetic. For instance, during removal of a distal aortic aneurysm, unavoidable division of lumbar arteries may deprive the spinal cord of its supplementary blood supply on which it may be particularly dependent because of the attenuated character of the anterior and posterior spinal arteries in the region, and paraplegia

may follow. In such an event, suspicion could fall on sub-arachnoid or epidural spinal block if either has been employed.

As another example, circulatory collapse frequently associated with cholecystectomy and formerly attributed to some vague intrinsic factor in patients suffering from gall-bladder disease is usually the result of occlusion of the inferior vena cava by abdominal packs or by stretching of that vessel over the vertebral column if the latter is extended for better access—a mechanism which may be facilitated by the use of muscle relaxants.

Apart from massive uncontrollable haemorrhage, pulmonary embolism is the gravest incident that can occur during surgical or obstetrical operations. It is tempting to regard such an accident as fortuitous. Occasionally this may be so, but careful consideration of the clinical findings will show that preventive measures are often possible. Although unrelated to the anaesthetic procedure, pulmonary embolism must be a matter of some concern to the anaesthetist. Having a share in the care and preoperative preparation of the patient, he should keep in mind the pathological conditions and the circumstances in which pulmonary embolism may occur and if necessary broach the possibility to his surgical colleague. In a period of thirty years I have seen eight patients die from pulmonary embolism during manipulation and reduction of fractured neck of the femur at the sixth to tenth day after the accident. Trauma and restriction of movement initiate venous thrombosis in the neighbourhood of the fracture and during manipulation, after an interval of some days, a thrombus may more readily be dislodged. There would, therefore, appear to be a danger period which might be avoided.

Pulmonary embolic block most commonly follows thrombosis of the deep veins of the pelvis or legs and in appropriate cases clinical evidence, for example, increased girth of a leg due to venous obstruction, should be looked for preoperatively or on successive days after operation. Other common sources of blood-formed pulmonary emboli are the right atrium and ventricle, the placental site, and the prostatic bed.

Pulmonary arteries may also be blocked by fat globules from the marrow of fractured long bones, tumour—such as hypernephroma involving the inferior vena cava—air sucked into a large vein, and, perhaps most tragic of all causes, glutinous amniotic fluid from the uterus during parturition.

The rapidly developing signs of acute cor pulmonale can be quickly recognized by the anaesthetist and when the arterial block is massive there is little that can be done quickly enough to prevent a fatal termination. It should be realized, however, that even a small embolus may produce a critical clinical state disproportionate to the size of the lesion, as a result of generalized reflex spasm of the pulmonary vessels and of the bronchi and coronary arteries. It is worth while, therefore, to attempt to eliminate these concomitant factors by the administration of spasmolytic agents.

Time does not permit consideration of other surgical and medical conditions of which anaesthetists should keep themselves well-informed, for example, meteorism which frequently jeopardizes the recovery of patients immobilized after operations on the hip and may wrongly be attributed to new anaesthetic

agents or methods. In Canada, particularly, I might have ventured to discuss with becoming respect the problem of stress and the positive contribution which, I believe, anaesthetists can make towards its prevention or alleviation in extensive surgical operations.

I hope I have indicated the significant overlap of interests between anaesthetic practice and medicine and surgery and the comprehensiveness of a career in our speciality. No longer need there be any inferiority complex in respect to it and I trust that all of us, especially the younger generation, appreciate the finely poised position we have reached in being accorded a responsible and respected share in the care of patients which I believe is adequate compensation for not having patients of our very own. In consolidating this responsible status and working harmoniously with our surgical, medical, and obstetrical colleagues, we can enjoy an acceptable executive and advisory function and, indeed, the best of two worlds.

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A STUDY OF METHOXYFLURANE ANAESTHESIA

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DURING ANAESTHESIA with methoxyflurane (1,1-difluoro 2,2-dichloro ethyl methyl ether: CH_3-O-CF_2CHCl_2), hypotension occurs as depth increases,[1] while arfonad infusion is said to have a greater effect than with other anaesthetic agents.[2] These and other clinical observations have stimulated this brief report, which concerns an assessment of sympatho-adrenal and other responses during a steady state of methoxyflurane anaesthesia in dogs, and during subsequent periods of hypercarbia and haemorrhagic hypotension.

METHOD

Eight dogs (average weight about 10 kg.) were anaesthetized with 2.5 per cent thiopental and the trachea was intubated with a no. 10 cuffed Magill tube, which was connected via a Ruben non-rebreathing valve to a 10-L. flow of 100 per cent oxygen. Two animals were allowed to breathe spontaneously throughout the entire study; the remaining six were ventilated with a Bird respirator (Marks 4 and 8). A femoral artery was cannulated for collection of blood samples and recording of blood pressure (Statham transducer and Sanborn recorder). Heparin (Connaught Labs. 1,000 units/ml.) was given to all dogs in an initial dose of 2 mg./kg., supplemented by 1 mg./kg. in three studies.

After the initial period of breathing 100 per cent oxygen (the average total thiopental dosage being 23 mg./kg.) control blood samples were taken and the oxygen flow was directed through methoxyflurane in a Boyle's ether bottle, adjusted to give average concentrations of approximately 0.6 per cent (range 0.55 to 0.78 per cent). Blood samples were withdrawn 30, 60, and 120 minutes after starting methoxyflurane administration. Thereafter, in five experiments (two with spontaneous respiration and three with controlled ventilation), 20 per cent carbon dioxide in oxygen was used as the vehicle for vaporizing methoxyflurane, and further blood samples were collected after 30 and 60 minutes. In the remaining three studies, the dogs were subjected to a haemorrhage of approximately 20 ml./kg., and a second haemorrhage of 10-14 ml./kg., blood samples for assay being taken approximately 15 minutes after each haemorrhage.

Each blood sample included 35 ml. for plasma adrenaline and noradrenaline assay,[3] 2 ml. for blood glucose estimation,[4,5] and 8 ml. collected anaerobically for determination of pH and pCO_2 of whole blood and bicarbonate of *separated*

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TABLE I

DATA OBTAINED IN ONE EXPERIMENT DURING WHICH SPONTANEOUS RESPIRATION WAS ALLOWED TO PERSIST DURING ADMINISTRATION OF AN AVERAGE VAPOUR CONCENTRATION OF 0.61 PER CENT METHOXYFLURANE IN OXYGEN

Time (min.)	pH	pCO_2 (mm. Hg)	HCO_3^- (mM./l.)	Adrenaline (μg./L.)	Noradrenaline (μg./L.)	Glucose (mg./100 ml.)	Arterial blood pressure systolic (mm. Hg)	Arterial blood pressure diastolic (mm. Hg)	Heart rate
Control	7.54	24	20	0.45	0.0	68	140	78	65
+30	7.16	74	23	0.56	1.4	71	128	81	174
+60	7.07	112	26	0.03	0.57	90	116	66	160
+120	7.00	135	24	0.21	0.66	128	142	78	163

TABLE II

AVERAGED DATA DURING 120 MINUTES OF STEADY STATE METHOXYFLURANE ANAESTHESIA (7 EXPERIMENTS), AND AS A RESULT OF SUBSEQUENT HYPERCARBIA (4 EXPERIMENTS) AND HAEMORRHAGE (3 EXPERIMENTS)

	Time (min.)	pH	pCO_2 (mm. Hg)	HCO_3^- (mM./l.)	Adrenaline (μg/L.)	Noradrenaline (μg/L.)	Glucose (mg./100 ml.)	Arterial blood pressure systolic (mm. Hg)	Arterial blood pressure diastolic (mm. Hg)	Heart rate
Steady State Ventilation	Control	7.51	28	22	0.08	0.19	75	155	90	119
	+30	7.46	32	22	0.14	0.25	72	116	71	119
	+60	7.45	33	21	0.28	0.22	74	107	67	122
	+120	7.42	34	21	0.08	0.26	72	97	63	118
Hypercarbia	+30	6.86	173	25	0.74	0.58	127	88	35	119
	+60	6.83	193	26	1.4	0.93	128	91	41	118
Haemorrhage	+20	7.39	27	16	0.50	0.15	96	44	24	123
	+40	7.32	32	15	2.2	0.51	86	37	16	154

plasma at a pCO_2 of 40 mm. Hg.[6] After each sample an equal volume of normal saline was injected intravascularly.

Student's t-test was employed for statistical analysis of the data.

Results

When these studies were begun, the intention was to assess some effects of methoxyflurane during both spontaneous and controlled respiration, but in one early experiment respiratory depression was so pronounced that progressive severe carbon dioxide accumulation occurred as anaesthesia continued (Table I). In the dog, methoxyflurane is clearly a potent respiratory depressant in concentrations below 1 per cent. Subsequent experiments were therefore conducted with controlled respiration. Muscle relaxants were not required to prevent spontaneous respiratory movements during ventillation with methoxyflurane.

Table II presents the average data obtained from seven experiments, and shows that during a steady state of methoxyflurane anaesthesia lasting for two hours, within an average arterial pCO_2 range of 25–35 mm. Hg, no consistent or statistically significant changes in plasma adrenaline or noradrenaline could be detected. The acute respiratory acidosis induced by ventilation with 20 per cent carbon dioxide in oxygen during methoxyflurane anaesthesia, evidenced by the lowered pH and increased pCO_2, caused consistent increases in plasma catecholamine concentrations. The rise in plasma adrenaline, from 0.15μg./L. at a pCO_2 of 32 mm. Hg to 0.74 at a pCO_2 of 173 mm. Hg (30 min.), and to 1.4 μg./L. at a pCO_2 of 193 mm. Hg (60 min.), was highly significant ($p<0.01$). Plasma noradranline increased significantly ($p<0.05$) from 0.23 μg./L. to 0.58 μg./L. after 30 min., and increased further to 0.93 μg./L. after 60 min. (not significant statistically because of the wider range of values measured).

As a result of haemorrhage (three experiments), highly significant increases in plasma adrenaline occurred ($p<0.01$), from 0.15 μg./L. to 0.50 and 2.2 μg./L. after the first and second haemorrhages respectively. The smaller increase in plasma noradrenaline evident after the second bleeding was not significant. Haemorrhage induced hypotension, tachycardia, and a metabolic acidosis.

Little change in blood glucose occurred during the 120 minutes of uncomplicated methoxyflurane anaesthesia, but hypercarbia was accompanied by highly significant rises ($p<0.01$). Blood glucose also increased as a result of haemorrhage, but not significantly.

Mean arterial pressure decreased gradually during methoxyflurane anaesthesia, from an average control level of 110 mm. Hg to 74 mm. Hg after two hours, the greater change being in systolic pressure. Heart rate increased in five experiments and decreased in three. The early response to carbon dioxide was a consistent fall in diastolic pressure, with more variable changes in systolic pressure. After one hour of hypercarbia, mean arterial pressure was decreased to 57 mm. Hg, largely accounted for by the reduced diastolic pressure.

In two initial studies, spontaneous respiration was allowed to continue during methoxyflurane administration. In one of these, arterial pCO_2 increased only moderately, to a maximum of 55 mm. Hg, and the findings are included in the

average data shown in Table II. In the other experiment, which is excluded from the averaged results, the respiratory rate after two hours of 0.61 per cent methoxyflurane was only 2 per min. at an arterial pCO_2 of 135 mm. Hg (Table I). In this study plasma noradrenaline concentrations were consistently higher than in any of the other seven experiments, although the early rise from a zero level at a pCO_2 of 24 mm. Hg to 1.4 μg/L. at a pCO_2 of 74 mm. Hg was not fully maintained thereafter.

No cardiac arrhythmias were noted on the blood pressure record during methoxyflurane administration. In one study an arrhythmia which was present after intubation was briefly accentuated when an abrupt increase in heart rate occurred as methoxyflurane was started, but a normal rhythm quickly followed. In no study were cardiac arrhythmias observed during the period of hypercarbia.

Discussion

These studies show that in the dog plasma adrenaline and noradrenaline levels are not significantly increased during anaesthesia with methoxyflurane (*average* concentrations 0.55–0.78 per cent in oxygen). In this respect, therefore, methoxyflurane resembles halothane,[7] but differs from diethyl ether, which in this laboratory has been found to induce consistent rises in plasma adrenaline and noradrenaline in dogs.[8]

The increased plasma catecholamine levels measured as a result of hypercarbia and haemorrhage during methoxyflurane anaesthesia show that sympatho-adrenal responses are not completely blocked by this agent when given in the concentrations employed for this study; these concentrations produced marked respiratory depression if spontaneous respiration was permitted. In this regard, methoxyflurane is similar to other anaesthetic agents, none of which appears to prevent catecholamine liberation from adrenal medulla and sympathetic nerves in response to the stresses of hypercarbia and haemorrhage.[8] It should be noted, however, that the increases in total plasma catecholamine level resulting from haemorrhage or hypercarbia during methoxyflurane anaesthesia were less than those previously measured during anaesthesia with halothane.[7] This could be interpreted as an indication of greater suppression of central or peripheral reflex sympatho-adrenal responses with methoxyflurane than with halothane, although for an accurate comparison of the two agents further studies would be required. It can only be stated that if some degree of sympatho-adrenal inhibition does occur during methoxyflurane anaesthesia, then the effect is incomplete.

Average mean arterial blood pressure levels before and after 120 minutes of methoxyflurane anaesthesia were almost identical to those measured in the similar study on dogs anaesthetized with 2 per cent halothane.[7] The consistent fall in diastolic pressure in response to hypercarbia was also common to both studies. During "steady state" ventilation, consistent bradycardia occurred with halothane but not with methoxyflurane. During halothane anaesthesia the heart rate increased as a result of hypercarbia to levels similar to those which were maintained before and during the hypercarbic state in methoxyflurane anaesthesia.

The stable blood glucose levels during 120 minutes of methoxyflurane anaesthesia and the increases with hypercarbia and haemorrhage correlate well with the plasma catecholamine concentrations. Wasmuth *et al.*[9] found an increase in blood glucose during clinical anaesthesia with methoxyflurane. Studies are obviously required in man, but it seems clear that the increased plasma catecholamine and blood glucose levels induced by diethyl ether in the dog are not seen with methoxyflurane. The severe respiratory depression produced by methoxyflurane in dogs apparently does not occur to the same degree in man. This interesting species difference merits further investigation.

Summary

Methoxyflurane anaesthesia in dogs produced respiratory depression, a gradual moderate decline in blood pressure with minimal changes in heart rate, and no significant effect on blood glucose or plasma adrenaline and noradrenaline levels. Hypercarbia induced an immediate hypotensive response and caused significant increases in both catecholamines; haemorrhage was followed by significant increases in plasma adrenaline. Blood glucose increased as a result of hypercarbia and haemorrhage during methoxyflurane anaesthesia.

Acknowledgments

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Résumé

L'anesthésie au méthoxyflurane, chez les chiens, s'accompagne d'une dépression respiratoire, d'une chute modérée graduelle de la tension artérielle avec des changements minimes du rythme cardiaque mais elle demeure sans effet important sur le taux de glucose sanguin, pas plus que sur le taux d'adrénaline et de noradrénaline plasmatiques. L'hypercarbie a entraîné une hypotension immédiate et a produit une augmentation importante des catecholamines; l'hémorragie a entraîné une augmentation importante du taux d'adrénaline plasmatique. Au cours de l'anesthésie au méthoxyflurane, à cause de l'hypercarbie et de l'hémorragie, le taux deglucose sanguin a augmenté.

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CLINICAL EVALUATION OF CERVICOTHORACIC SYMPATHETIC NERVE BLOCK IN THE MANAGEMENT OF BURNS OF THE HANDS*

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IN LESIONS OF THE HAND, the imperative therapeutic object is to obtain not only recovery, but an early and complete recovery such as to avoid scars, painful and vascular disturbances, muscular and joint impairments or dysfunctions. The cardinal factors for proper therapy of injuries of the hand are: (*i*) rigorous asepsis, (*ii*) earliest possible mobilization, (*iii*) intensive antalgic treatment and maintenance of good circulation in the affected limb, so as to avoid any possible post-traumatic reflex algodysthropy.

A therapeutic programme of this type is required especially for the treatment of burns of the hand. It is known, in fact, that the first consequence of a thermal injury is vasoparalysis and oedema of the injured region. Furthermore, when the burn involves deep epidermal layers, it also affects the pain nerve endings, causing a tumultuous reflex reaction, which locally causes a vasospasm in the marginal zones of the burned area in which vasoparesis has not yet occurred. A circulatory decompensation occurs, which extends beyond the boundaries of the areas affected by the thermal injury with circulatory stasis in some areas and impaired circulation due to vasospasm in others. The ultimate consequence of this condition is tissue hypoxia, which, in turn, becomes another source of painful stimuli that can aggravate the pain and circulatory decompensation. A vicious circle is thus formed: pain, vasospasm, aggravation of the pain, and vasospasm, which usually gives rise to a greater anatomic injury than that caused by the initial thermal stimulus.[1] An effective treatment of pain, therefore, has primary therapeutic importance, being capable not only of relieving the painful symptoms, but also of rapidly preventing the formation of the above mentioned reflex arc, thus avoiding extension and aggravation of the primary lesion.

Of the many antalgic measures reported in the literature, the most noteworthy seems to be regional nerve block, referred both to somatic sensibility and sympathetic innervation. The observations which are the subject of this paper have preferably turned to this method.

This series includes 140 cases, 70 of which were treated with nerve block, and 70 who received other antalgic treatment. The second group is considered as a control group with respect to the first. Though it was evidently impossible for the lesions to be identical in every case in this series, the patients were selected so as to satisfy a number of requirements.

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(1) Extension of burns was never more than the conventional index of 2.5 per cent, corresponding approximately to the hand and forearm.

(2) Depth of lesion was never the conventional limits of the third degree.

(3) First degree lesions were also excluded when, at the time of hospitalization, pain was no longer objectively demonstrable.

(4) Only adult patients were included.

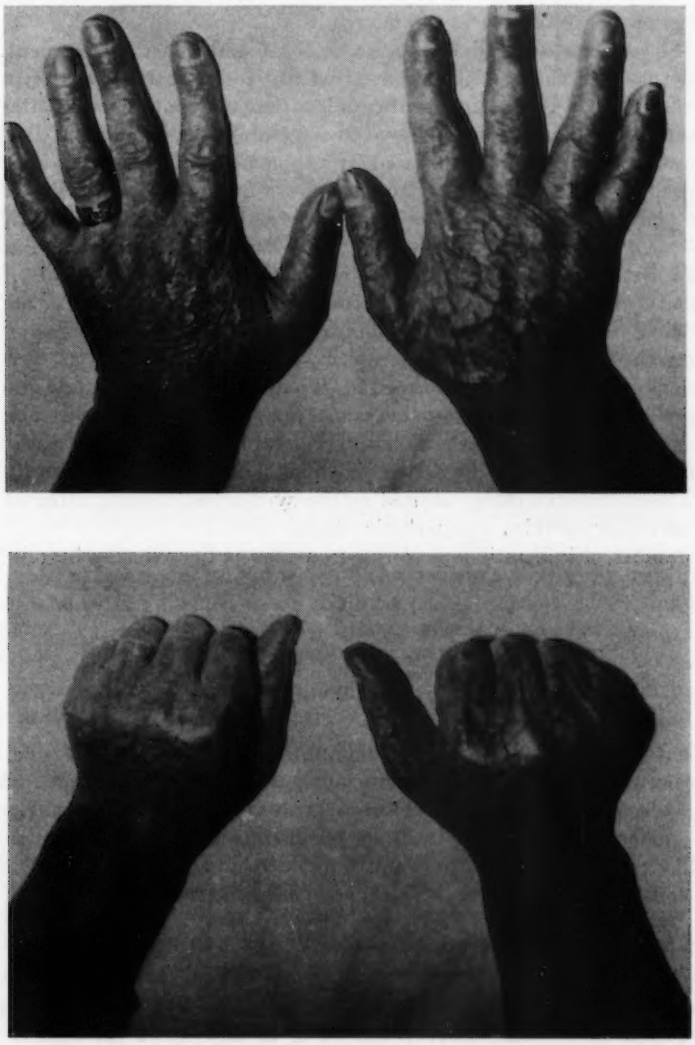

FIGURE 1. Both hands similarly burned. Left side received cervicothoracic sympathetic nerve block.

(5) All patients who received nerve block treatment, but in whom open treatment of the burn could not be performed according to the standard procedure for the cases examined, were excluded.

Figure 1 shows the case of a patient belonging to both groups of this series. Both upper limbs showed burns of the same extent and severity. However, nerve block was limited to the left arm, and the right arm was used as a control with respect to the left one; in ideal conditions, therefore, for comparative evaluation of results, as clearly shown in the figure.

All 140 patients were given the same local and general treatment. Local therapy was as follows: rigorous and extensive disinfection of the marginal tissues of the lesion (burns are aseptic, unless they are contaminated after injury by non-sterile medical dressing or by other factors), free exposure of the burned limb in aseptic surroundings, isolated by means of sterile bandages and towels. The patient was kept in a room containing an ozonizer, and having sufficient relative humidity to avoid raising dust. General therapy consisted of slow drip infusion of a 5 per cent dextrose plus saline solution in sufficient amount to maintain the salt and water balance in relation to the daily oral fluid intake, to the urinary excretion, and to the amount lost by perspiration. Plasma transfusions were required in a limited number of cases with more extensive loss of fluid from the injured part.

Nerve block therapy was performed in 70 patients as follows:[1] upon admission: brachial plexus block via the supra-clavicular approach associated with infiltration of the cervicothoracic sympathetic chain, preferably by the anterior paratracheal approach, using 0.1 per cent pontocaine plus 1/250,000 epinephrine; after 12 hours: a second nerve block, limited to the sympathetic chain; after 24 hours: (when necessary) a third sympathetic block.

The number of infiltrations performed on each patient during hospitalization is shown below.

Total number of infiltrations	70
Brachial plus stellate ganglion block	23 (32.8 per cent)
Second stellate ganglion block	35 (50 per cent)
Third stellate ganglion block	12 (17.1 per cent)

In the remaining 70 patients (control group) antalgic treatment was performed by parenteral administration of narcotic analgesic drugs as follows: pentobarbital 100 mg., promethazine 50 mg., chlorpromazine 50 mg., repeated alternatively every eight hours. In order to evaluate the effectiveness of somatic and sympathetic block for antalgic treatment of burns of the hand, four clinical features will be considered separately, comparing them in the two groups.

Clinical Recovery Time

Times required for clinical recovery are shown in Table I. In patients receiving antalgic treatment by administration of narcotic analgesic drugs, the average time required for clinical recovery was 14 to 20 days (74.2 per cent of cases). This corresponds to the figures reported in the literature: three weeks in Thalheimer's series of second-degree burns;[2] 15 to 20 days in Viganovi's series of

TABLE I

CLINICAL RECOVERY IN 70 CASES

	Patients treated with nerve block	Untreated patients
Clinical recovery		
7 days	8 (11.4%)	
10 days	43 (61.4%)	13 (18.5%)
14 days	19 (27.1%)	25 (35.7%)
20 days		27 (38.5%)
Over 20 days		5 (7.1%)
Complications		
Local		3 (4.2%)
Visceral	3 (4.2%)	14 (20%)

second-degree burns;[3] 15 days in Edouard's series of second-degree burns treated by cryotherapy;[4] 14 to 18 days in the series of Blocker,[5] Taylor,[6] and Wallace,[7] in which open treatment of the lesion was given. Similar observations have also been reported by other authors.[8, 9, 10] On the other hand, in the group given nerve block treatment, the average time required for clinical recovery was 10 days (61.4 per cent of cases). No reports of more rapid recovery of the second-degree burns have been found.

Local Complications

Cicatrization of burns did not differ significantly in the two groups. In the first group, local oedema showed evidence of regression as early as 24 hours after the injury, whereas in the control group regression took place after about 36 or 48 hours, as generally reported in the literature.[6, 9] Reabsorption index was essentially the same in both groups of patients. All the 70 patients given analgesic block treatment were discharged when the lesions were completely re-epithelized, and the hand or the affected joints could be moved with ease, without pain, or vascular or cicatritial impairment. In three cases of the control group (4.2 per cent of cases) it was necessary to cover a granulation in the burned area. According to Rosselli[11] this plastic procedure should be used routinely in combined second- and third-degree burns, whereas Evans[12] states that lesions that do not go beyond the papillary state should heal spontaneously when treated with an open aspetic method.

Visceral Complications

In three of the 70 patients given analgesic block treatment, albuminuria occured on the second day (4.2 per cent of cases). In 14 of the 70 patients of the control group various visceral complications were noted (20 per cent of cases): renal block on the third day with recovery on the ninth day (one case); lesion of the bladder mucous membrane observed cystoscopically (one case); albuminuria and microscopic haematuria about the second and third day (12 cases). Body temperature remained more or less unchanged in 125 of the 140 patients. In the remaining 15 cases, all belonging to the control group, pyrexia was observed during the first hours after injury and disappeared about the forty-eighth hour.

Pain

Behaviour of the painful symptoms differed in the two groups of patients. Pain was promptly relieved immediately after brachial plexus block and the absence of painful symptoms persisted for a much longer time than might be expected in relation to the anaesthetic potency of the solution used. In reality, a second nerve block was never required before the twelfth hour, and stellate ganglion block alone was sufficient. Furthermore, a second antalgic treatment was required in only 35 of 70 patients (50 per cent of cases), whereas a third infiltration was required in only 12 patients (17.1 per cent of cases). It is also interesting to note that psychomotor agitation which is usually present in patients with burns and is generally attributed to a more or less latent shock disappeared as soon as a somatic nerve block was performed, whereas stellate ganglion block sufficed to eliminate residual pain, when present, in the twelfth hour or later. On the contrary, in the control group, continuous administration of narcotic analgesic drugs every eight hours was required. Considerably painful symptoms reappeared in these patients during the first 36 or 48 hours, as soon as the analgesic effect of the injected drug tended to decrease. It should finally be noted that the visceral complications mentioned above appeared in the patients with the severest subjective symptoms, in spite of scrupulous infusion and transfusion therapy.

Conclusions

These findings indicate that somatic and visceral nerve block can have considerable clinical effects in the treatment of benignant burns of the upper limbs. Its favourable effect seems to affect the time required for healing and the prevention of visceral complications. The effects of this method in the epithelization of the wounds seem less notable, as the types of burns taken into consideration show a good tendency to spontaneous healing, provided that aseptic conditions are maintained in the injured region. In effect, this process seems only accelerated by nerve block as compared with controls and not substantially modified in its anatomic and functional evolution.

Furthermore, nerve block therapy offers marked advantages over a more general antalgic treatment; the physiological injury to the patient by nerve infiltration, with the doses of anaesthetic used, seemed, in fact, by far smaller than the potential dangers of large doses of narcotic analgesic drugs such as are required to relieve the pain of burns of the hand. To underline the importance of this fact, the observations of Elman, [13] later confirmed by other authors,[14, 15, 16] should be stressed. According to these workers, mortality due to burns seems significantly greater following the administration of large doses of narcotic analgesic drugs. On the contrary, inherent dangers of nerve block therapy in experienced hands seem negligible. Only two cases of pneumothorax occurred in this series and healed properly without objective or subjective consequences to the patient.

Résumé

Un blocage nerveux somatique et viscéral peut entraîner des effets cliniques salutaires dans le traitement des brûlures bénignes du membre supérieur. Le

blocage semble produire comme effet adjuvant: une accélération de la guérison et une protection contre les complications. Les effets du blocage sur l'épithélisation des blessures sont moins évidents car les types de brûlures que nous avons étudiés ont montré une tendance spontanée à la guérison, à la condition que l'infection n'envahisse pas la région. De fait, la guérison semble accélérée par le blocage nerveux si l'on fait une comparaison avec des cas témoins et elle ne présente pas de modifications substantielles dans son évolution anatomique et fonctionnelle.

De plus, le blocage nerveux thérapeutique présente des avantages marqués sur un traitement antalgique plus général. Le dérangement physiologique produit chez le malade par l'infiltration, sachant les doses d'agent anesthésique employées, semble, de fait, bien infime comparé aux dangers possibles que pourraient constituer pour le malade de fortes doses d'analgésiques comme on est obligé d'en donner pour soulager les douleurs occasionnées par les brûlures de la main. Pour souligner l'importance de ce fait, nous citons les observations de Elman,[13] confirmées ultérieurement par d'autres auteurs.[14,15,16] D'après ces auteurs, le taux de mortalité à la suite de brûlures semble plus élevé si l'on fait usage de fortes doses de médicaments analgésiques. Par contre, dans des mains expérimentées, les risques inhérents au blocage nerveux thérapeutique semblent négligeables. Au cours de cette série, nous avons observé seulement deux cas de pneumothorax et ces deux complications sont demeurées sans conséquence objective ou subjective pour les malades.

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REMOTE CONTROL GENERAL ANAESTHESIA AND BRONCHOGRAPHIC TECHNIQUE IN INFANTS AND CHILDREN*

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IN THE PAST, a great variety of anaesthetic procedures, both local and general, have been introduced to meet the requirements of bronchography. The anaesthetist has had to meet the individual exigencies of the medical internist trained in endoscopy, the otolaryngologist, and sometimes the thoracic surgeon who performs his own diagnostic procedures. Under such circumstances it is not surprising that generally accepted standarized bronchographic and anaesthetic techniques have yet to be established and remain a challenge.

In adults, local anaesthesia is very adequate and acceptable to the patient provided the bronchographic procedure is performed by a skilled specialist. For obvious reasons, bronchography performed under local anaesthesia in children can be and usually is a formidable enterprise. With the possible exception of aerosol nebulization,[1] local anaesthetic procedures in children are not only difficult to perform but also expose them to toxic reactions. They are usually time consuming and require a great deal of patience. Notwithstanding complete local anaesthesia of the hypopharyngeal region and upper respiratory tract, the subsequent introduction of the dye into the bronchi can in itself be difficult to perform in children. Methods based on the fact that opaque material will be aspirated into the larynx rather than be swallowed after complete anaesthetization of the hypopharyngeal region,[2] usually require a large quantity of dye which is introduced via catheter into the nasopharyngeal region and exposes the patient to toxic reactions. The postural methods[3] subesquently used to distribute the dye by gravity are not always reliable. A bilateral study often cannot be performed in one session. The management of such a procedure proves to be very difficult and exhausting both for the diagnostician and the patient. The taking of films with a squirming, balky, half-drowned child, fighting for breath and who must be forcibly restrained, does not always give excellent results and necessarily exposes the hands of the attendants to a greater radiation hazard.

Techniques conducted under general anaesthesia for bronchography in children are numerous. An excellent review has been presented by Parkhouse[4] and there is no necessity to elaborate on classical techniques and their variations here. Essentially, these techniques are performed either with or without an endotracheal airway after establishment of anaesthesia, usually with ether.

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When the child is not intubated, the dye is injected by a syringe via a small catheter introduced through the mouth or nose into the trachea. Sometimes in the "drowning technique" as described under local anaesthesia, manipulations are reduced to a minimum and the opaque medium is poured directly into the mouth of the anaesthetized child. These procedures necessarily entail a serious risk to the child patient. In spite of the explosion hazard, moderately deep ether anaesthesia is usually recommended and after initial open ether induction there is no means to control the level of anaesthesia. Excessive amounts of opaque medium are required, sometimes as much as 30 c.c. in larger children.[4] Under such circumstances it cannot be denied that the child is subjected to a state which is somewhat similar to the process of drowning. Associated local anaesthesia of the upper respiratory tract does not sufficiently protect from bronchospasm. Respiratory obstruction and bouts of cyanosis are bound to occur too frequently. It follows that uncontrolled blind flooding of the bronchial system under general anaesthesia in a child who is not intubated necessitates frantic rapid manœuvres to obtain X-ray records in order to initiate resuscitation without undue delay. Such a procedure is not conducive to consistent good results and the upper lobes often present incomplete filling. Here again the anaesthetist is very likely to expose his hands to the primary beam of the X-rays while attempting to maintain a clear airway in his patient.

When bronchography is performed with the help of endotracheal intubation involving no special modification of anaesthetic instrumentation, the radio-opaque material may be injected into the bronchi from a syringe directly into the endotracheal tube or through a small catheter directed into the lumen of the endotracheal tube. The dye may also be injected through a small catheter, which either lies freely alongside the endotracheal tube, or is incorporated into the artificial airway. Here the first two methods necessitate interruption of anaesthesia while the injection procedure is carried out with the patient breathing spontaneously. No interruption of anaesthesia and oxygenation to the patient need occur in the last two methods and relaxant drugs may be used with controlled respiration throughout by means of a closed or non-rebreathing anaesthetic circuit. Although this category of techniques allows for better oxygenation and anaesthetic management, the injection of dye still remains an uncontrolled flooding procedure. The assistance of fluoroscopy does not appreciably compensate for deficiencies in anaesthetic and bronchographic techniques. With a free catheter, depending upon which technique is being used, its manipulation may be either difficult or time limited. An integrated endotracheal tube-catheter system does not lend itself to meticulous work. When a relaxant drug is not used, coughing may cause undesirable obliteration of bronchial detail by blowing dye into the alveoli. The anaesthetist's hands are still dangerously close to the direct X-ray beam and the explosion hazard is ever present should ether be used as the anaesthetic agent of choice.

Amongst the more recent methods, Smith[5] recommends the use of a Y-adapter attached to the endotracheal tube. One arm of the adapter is fitted with a perforated nipple through which a plastic catheter is passed for insertion of the opaque material. The other arm of the Y-adapter is connected to either a non-

rebreathing or circle system. In spite of the explosion hazard ether is used to avoid the risk of respiratory depression. Topical anaesthesia of the cords and trachea is associated. Ventilation is assisted or controlled during the procedure but apparently relaxant drugs are not used. The closed anaesthetic circuit is cumbersome for such a procedure in children. The non-rebreathing technique with either an Ayre's T-tube or a manually operated Stephen-Slater or Leigh valve dangerously exposes the anaesthetist's hands to radiation during assisted or controlled respiration. Bowering[6] presents a very similar procedure, but here a non-explosive type of anaesthesia with a relaxant drug is recommended. Respirations are controlled throughout the procedure. The anaesthetist actually participates in the bronchographic phase by causing the medium to be evenly spread under the influence of deep controlled respirations. It is claimed that the amount of medium used is minimized. The presentation is somewhat lacking in detail and precautions against the radiation hazard seem to be of little concern.

From the foregoing it becomes apparent that successful bronchography under general anaesthesia in children depends on a number of factors.

Improved anaesthesia for bronchography will necessarily result in better performance for the endoscopist and refinements in bronchographic technique will also contribute to better anaesthesia. Oversimplified anaesthetic techniques requiring a minimum of instrumentation do not sufficiently protect the patient nor do they provide adequate conditions to the endoscopist which are necessary to avoid a veritable drowning episode. This implies that the endoscopist should be provided with conditions that will allow him to perform his work meticulously and to limit the quantity of dye injected to a minimum in order to avoid respiratory obstruction and alveolar filling. Since coughing causes alveolar filling which contributes to cyanosis and also interferes with interpretation of X-ray records, the cough reflex must be completely eliminated by appropriate means. Since bronchography under fluoroscopic control must be carried out in total darkness, the endoscopist and anaesthetist must work in uncluttered surroundings. The anaesthetist must give his undivided attention to his patient and should eliminate from his technique unnecessary distracting manœuvres. Groping in the dark for instruments, drugs, and so on must be avoided. For these reasons, anaesthetic instrumentation must be designed in such a manner that will provide immediately available finger-tip controls, easily accessible for manipulation in the dark. Flowmeters on anaesthetic machines should be provided with luminous dials visible in the dark. The Boyle anaesthetic machine is so equipped. Under such working conditions, however, a subdued light source should be immediately available to the anaesthetist for intermittent observation of the child's colour.

Notwithstanding the overwhelming recommendations with regard to ether as the choice of anaesthetic agent for bronchography in children, we firmly believe that an explosive agent should not be used in the immediate vicinity of an X-ray machine. There are enough problems involved in this anaesthetic procedure without having to cope with the explosion hazard. Non-explosive anaesthetic agents associated with a relaxant drug can provide a well-controlled superficial degree of narcosis and a fast return of natural defense mechanisms after the procedure.

With improved bronchographic and anaesthetic techniques, the advantages of diagnostic bronchography can be extended to younger children and infants. This means that the endoscopist and anaesthetist will be called upon more often to perform this procedure and hence will be faced with the problem of repeated exposure to radiation.[7] Therefore not only should usual precautions be observed, such as the wearing of a leaded apron by all those attending the procedure, but also innovations should be incorporated in the anaesthetic technique so as to either nullify or at least diminish the radiation hazard to the unprotected hands. Herein lies the advantage of remote control anaesthesia whereby the hands of the anaesthetist need not be exposed to the useful X-ray beam. Added to these considerations is the fact that the more precise a bronchographic procedure becomes, the longer the performance time and hence the greater the radiation hazard.

In bronchography it is essential that precautions against the radiation hazard be observed as recommended for any other procedure involving fluoroscopy, and set forth in Handbook 60 of the National Bureau of Standards.[8] When unmodified techniques of general anaesthesia for bronchography in infants and children are used, our observations reveal that regardless of an honest attempt to apply fundamental safety precautions, the radiation hazard to the endoscopist and anaesthetist is greater than in other procedures where there is no necessity for the hands of the operators to be in close proximity to the useful X-ray beam. As a general rule, the hands, either bared or gloved, should never encroach upon the direct path of the useful X-ray beam unless the beam is attenuated by the patient. In bronchography the wearing of gloves by the endoscopist and anaesthetist greatly hamper manipulations and they must proceed with their bared hands. By restricting the field size to a minimum, a skilled radiologist can safeguard the hands of his colleagues from the useful X-ray beam during fluoroscopy. Spot filming and the recording of standard size films, however, necessitate a wider beam which infringes on a greater area than the fluoroscopic control beam. This introduces another hazard to the hands of the operators which is difficult to control but which nevertheless should not be ignored.

When the hands are held at a distance of 18 inches from the periphery of the X-ray beam, they receive from five to ten times less radiation in relation to the maximum scattered dose. By increasing this distance one foot more, scattered radiation decreases by a negligible factor of 1.5 to 2. We therefore recommend as a safety precaution that the hands of the operators during bronchography be kept at a distance of not less than one and one-half feet from the periphery of the X-ray beam covering the widest field.

The technique developed by us was arrived at by joint consideration of all the problems involved. Requirements for the safety of the patient have always been foremost in our considerations and we have attempted to fulfill as well as possible all other requirements previously discussed.

The principle involving the injection of dye by means of a catheter passed through the endotracheal tube without interfering with the simultaneous maintenance of general anaesthesia and artificial respiration is attributed to Baker[9] and has been adapted to our technique. In Baker's method of bronchography in

children under general anaesthesia, the catheter is not inserted beyond the bifurcation of the trachea and the injected dye is dropped in either bronchus by gravity through suitable posturing of the patient. Ether is used and no special precaution to reduce the radiation hazard is recommended. Although the principle of this method was introduced in 1941, it is surprising to find that so many attempts have since been made at more simple procedures which have ignored the basic and logical technical recommendations set forth by Baker. The objection has been raised that a catheter inserted into the narrow lumen of a small endotracheal tube, such as is used in infants, restricts the airway and causes partial obstruction.[10] For this reason, we find that the use of an endotracheal tube in infants and children is excluded from many techniques. The contention is certainly justified when a large quantity of radio-opaque medium is used, which by itself can cause severe mechanical obstruction. We are convinced, however, that a capillary polyethylene catheter small enough to allow free ventilation between its wall and that of the endotracheal tube can always be found, however small the lumen of the endotracheal tube. A thin-walled endotracheal tube should be used. The quantity of dye injected should be restricted to a minimum compatible with the size of the patient. This will eliminate a source of gross respiratory obstruction and will not interfere with results. Intermittent positive pressure ventilation is also an important compensatory factor for the resistance which may result from the introduction of a small catheter into the lumen of the endotracheal tube. It may be argued that free injection of an opaque substance through a capillary-sized catheter is impossible. The same law applies to small bore catheters as it does for small needles[11] so that it is always possible to inject the medium by using a small capacity syringe which provides a greater mechanical advantage. Preheating of the contrast medium reduces viscosity and facilitates injection.

The self-contained anaesthetic apparatus is illustrated in Figure 1. It consists of a non-rebreathing, semi-automatic Fink valve[12] mounted on a metallic extension tube 18 in. long. An oral right-angled Adams' connector provided with a suction orifice is mounted on the distal extremity of the valve. The uncapped suction orifice is fitted with a short length of rubber tubing $1\frac{3}{4}$ in. long. This tube is inserted through a tight-fitting circular aperture in a specially designed clamping device provided with a dual kinking and constricting action. This clamp mechanism is relayed to a thumb activated remote-control lever by a Bowden cable. The proximal extremity of the tubular extension is mounted with a reservoir bag designed by Ruben.[13] The Ruben bag is a self-inflating device fitted with an air intake valve. It has been modified to administer an anaesthetic mixture by incorporating a perforated plastic chamber on the air intake valve. This perforated chamber bears a nipple which receives a delivery tube from the anaesthetic machine. Thus gases are delivered to the bag during the expansion phase only and excess flow escapes to the atmosphere, by the application of Ayre's insufflation principle[14] in order to avoid pressure build-up from this source. The Ruben bag is manufactured in two models. One model is provided with an oxygen inlet nipple incorporating a check valve. This inlet nipple presents a pressure build-up hazard in our apparatus when used for the delivery of gases.

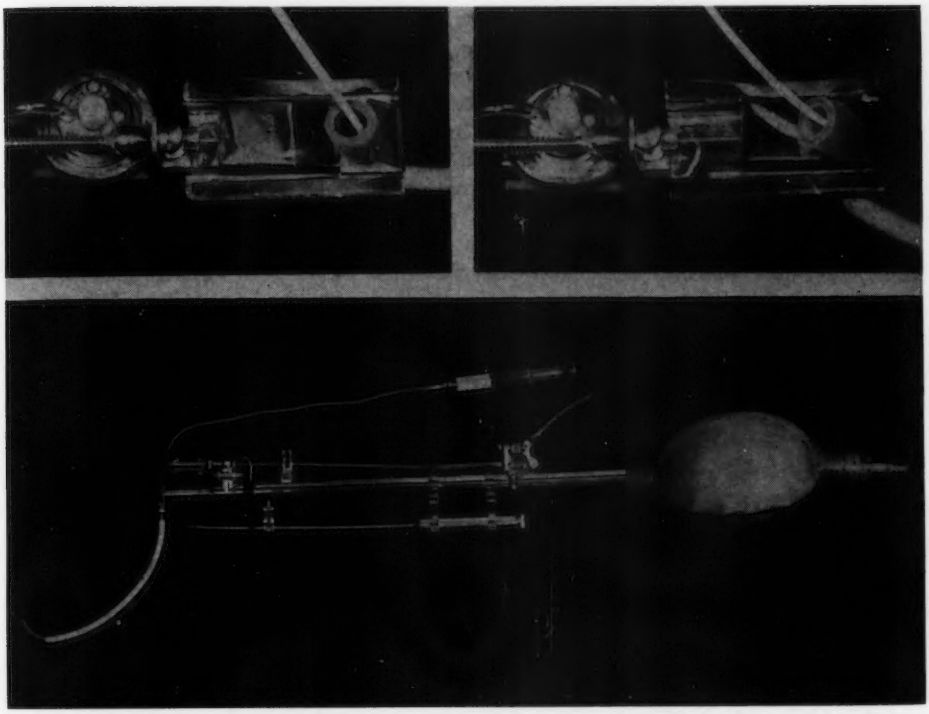

FIGURE 1. Remote control anaesthesia apparatus for bronchography showing injecting catheter connected to 10 c.c. syringe and passing through endotracheal tube. Left insert shows clamp mechanism operated by remote control in the open position. Right insert shows clamp in the closed position (see text for description).

We recommend the model which is manufactured without an oxygen intake nipple and modified according to our specifications for gas intake. A flashlight extension powered by a pen-type battery is mounted as shown on the long tube. This immediately available light source is intended for intermittent observation of the patient's colour and can be turned on and off at the proximal end of the tube.

In use, the pressure release valve on the Fink valve is kept closed at all times. Depression of the thumb lever causes the rubber tubing, through which various size catheters may be introduced, to be sealed hermetically around the catheter by the clamp, thus allowing the anaesthetist to control respirations. The clamp is momentarily released to allow the endoscopist to insert and properly locate the catheter inside the bronchus. The clamp must also be momentarily released during injection of the opaque material. To deliver the anaesthetic mixture to the patient or to administer oxygen, the clamp is closed and controlled respiration is performed by manual intermittent pressure on the Ruben bag. As mentioned before, pressure build-up cannot possibly result from a high flow of gases, although the pressure release on the Fink valve is kept closed.

The Ruben bag is an indispensable part of the apparatus since it releases the anaesthetist from the obligation of periodic adjustment of gas flows following initial setting to meet the variable volumetric ventilatory demands of the patient. Should gas delivery be insufficient to completely occupy the volume of the self-inflating bag, air dilution will occur through the air-intake valve in the bag. Excessive air dilution can be prevented by limiting the rate of inflation of the bag with the hand. Thus the anaesthetist is free to give his undivided attention to his patient, an essential part of the procedure. All that is required of him is an occasional glance at the luminous flowmeter dial to ascertain that gas flow settings have not been accidentally disturbed.

The tubular extension of the apparatus is intended as a safeguard against the radiation hazard. Unmodified anaesthetic apparatus necessarily exposes the anaesthetist to this danger. This is particularly true in infants and small children where the lower part of the head almost encroaches upon the useful beam of the X-rays. With a standard manual non-rebreathing technique, the anaesthetist's hands necessarily come dangerously close to the beam should controlled respiration be required. This manœuvre entails simultaneous manual obstruction of the expiratory valve with each compression of the reservoir bag. In the closed circuit technique, the drag of the cumbersome breathing tubes must be supported at a point close to the endotracheal tube to prevent accidental extubation.

The entire apparatus may be quickly and completely dismantled for cleaning and sterilizing. This necessary procedure is extremely important in infected cases.

Prior to the bronchographic procedure, postural drainage is carried out if excessive secretions are present. When the patient is ready, a fairly heavy premedication compatible with age, weight, and general condition is given. Should the child not be sufficiently sedated when he arrives for his diagnostic procedure, we refrain from aggressive induction of anaesthesia and supplement the premediation with rectal pentothal. A short 22- or 23-gauge needle is installed intravenously either in the arm or the foot and an isotonic glucose solution is administered from a bottle containing a small quantity of fluid to avoid accidental cardiovascular overload. The needle is never inserted under duress and if necessary it is inserted after induction of light fluothane narcosis by the non-rebreathing technique. A small bore intravenous extension set with a readily accessible injection site is installed for the administration of drugs during the procedure.

If necessary, prior to the bronchography, bronchoscopy is carried out under general anaesthesia. The same precautions as recommended for bronchography are observed during this preliminary procedure.

Endotracheal intubation is performed after induction with fluothane or a small dose of pentothal intravenously followed by an appropriate dose of succinylcholine.

Local spray of the vocal cords and trachea can be a hazard in children and it is best to avoid it if at all possible. On standing, local anaesthetics for topical anaesthesia have the property of crystallizing out of solution as a result of evaporation of the solvent and the crystals accumulate along the distal end of the atomizer extension tube and around the nozzle. The solution which remains in the tube may become highly concentrated also through evaporation of the

solvent. One single spray from such an atomizer which has been standing overnight or longer after previous use can vehicle not only a strongly concentrated solution but also undissolved crystals of the anaesthetic agent. In children the best precaution is to limit topical anaesthesia of the vocal cords and trachea to a lubricating anaesthetic jelly applied to the endotracheal tube.

Following intubation, tracheal and bronchial suction is carried out by the anaesthetist if necessary by means of a small suction catheter passed through the endotracheal tube. The endotracheal tube is then connected to the remote control apparatus and anaesthesia is maintained by a non-explosive mixture of oxygen, nitrous oxide, and fluothane. For this combined insufflation and inhalation non-rebreathing technique, the total flow of gases is set at around 8 L. per minute and the oxygen concentration is established at 40 per cent or higher.

Control of the cough reflex is obtained by complete curarization either with a continuous intravenous succinylcholine drip or by intermittent administration of the relaxant drug.

Controlled respiration is carried out by compression of the Ruben bag with the clamp closed around the catheter inlet tube. As explained previously, should air dilution occur during controlled respiration, the potency of fluothane is such that this will not appreciably disturb the degree of narcosis. Owing to smaller volumetric ventilatory requirements, air dilution is less likely to occur in infants and children than in adult patients. Moreover, deep anaesthesia is not required when a relaxant drug is used. The clamp is now released momentarily to allow the endoscopist to introduce a polyethylene catheter of appropriate size, previously filled with pre-heated opaque medium, into the endotracheal tube.

The endobronchial opacification technique in infants and children derives from the adaptation of principles which govern a method for preliminary selective segmental bronchography, usually performed under local anaesthesia in adults. This method was recently described by one of us (A. M.) and includes a report of 335 cases.[15]

Preliminary isolated segmental bronchography in adults is achieved by the use of endobronchial injecting catheters specially designed for this purpose by Metras[16] in 1947. Under fluoroscopic control, Metras catheters of proper size and possessing appropriate molded tips are alternately inserted into the corresponding lung, lobe, and segment. Should the radiologist observe segmental pathology while the opacification procedure is in progress, extemporaneous records are made on spot films after which the bronchogram is completed and standard postero-anterior lateral and oblique films are recorded. This procedure applies in reverse a bronchographic technique described by Nordenström[17] whereby diffuse bronchial instillation is initially attempted after which the segments not injected are filled selectively. The extemporaneous recording of what appears to be segmental pathology under fluoroscopic observation presents a definite advantage. Loss of detail which may otherwise result from subsequent bronchial emptying is thereby avoided.

Although Metras catheters of small calibre are not available at present, the foregoing meticulous preliminary selective procedure may be attempted under general anaesthesia in infants and children by the substitution of polyethylene

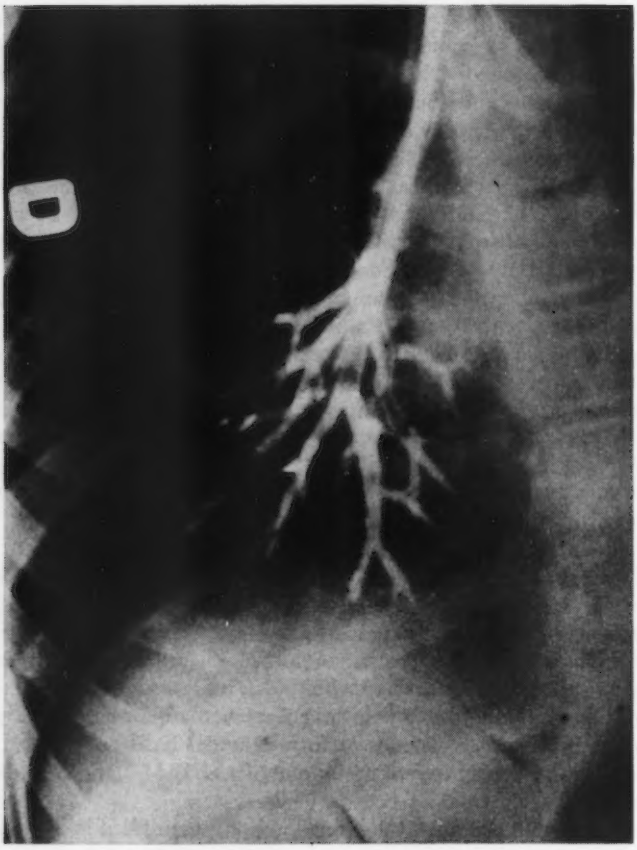

FIGURE 2. This right lower lobe bronchogram of a six-year-old child obtained under general anaesthesia illustrates possibility of preliminary selective lobar bronchography in children.

capillary tubing for the Metras catheters. Obviously, with these improvised injecting catheters, preliminary isolated segmental selectivity cannot be obtained in infants and children. Preliminary selective opacification of lobar dimension, however, may be obtained (Figs. 2 and 3). Under fluoroscopic control, the endoscopist localizes the polyethylene tubing, previously opacified by filling with radio-opaque medium, at the level of the suspected segmental pathology. Perfect co-ordination and timing must prevail between endoscopist and radiologist who must not be distracted by having to worry over the condition of the patient. Manipulation of the catheter must be carried out blindly while its position is checked by intermittent fluoroscopic observations of short duration, during which time the endoscopist must remove his hands from the direct X-ray beam. In order to provide adequate protection to the endoscopist from the radiation

FIGURE 3. Same patient as in Figure 2 showing completed right bronchogram in the lateral position.

hazard, the injecting catheter should be at least two feet long. Whenever necessary, the anaesthetist ventilates the patient by closure of the clamp on the remote control anaesthetic extension and the condition of the patient is checked by momentarily flashing on the extension light. Advantage is taken of the intermittent fluoroscopic images to check on cardiac action and the effectiveness of artificial ventilation. When the catheter has been properly placed, the endoscopist proceeds to a controlled injection of a discrete quantity of radio-opaque medium. The amount of dye required for adequate double contrast pictures is determined by the radiologist. Simultaneously, the anaesthetist applies gentle positive pressure by intermittent compression of the Ruben bag. At this particular time, partial closure of the remote control clamp without causing constriction of the injecting catheter will not interfere with either the injection procedure or the control of respiration. Thus under fluoroscopic and ventilatory control the

FIGURE 4. Spot film illustrates position of opacified plastic injecting catheter in the left main bronchus of a fourteen-day-old infant. Note small size catheter (no. 5 Fr.). Endotracheal tube (no. 0 Magill scale) may be discerned in the trachea and it may be seen that its lumen is not grossly reduced by the presence of the small injecting catheter.

minimum quantity of radio-opaque medium required to coat the bronchi under observation is evenly spread into the lung segments which are opposite the site of the catheter tip.[6] Under complete apnoea, spot films are made the moment pathology is detected and also following complete filling of the lobe under observation. Taking advantage of the natural curvature of the plastic catheter, the tip is again oriented towards another lobe and the same procedure is again repeated. After complete opacification of the bronchial arborization of the lung under examination, standard films are made of the completely immobilized lung.

After the procedure, following return of adequate spontaneous respiratory activity, the X-ray table is tilted in the head-down position to accomplish gravity

FIGURE 5. Same patient as in Figure 4. Right bronchogram showing Dionosyl® in right main bronchus and demonstrating patency of the roots of the segmental bronchi. Note injecting catheter in right main bronchus. Here intentional forceful injection of Dionosyl has caused some spilling of the opaque medium into the left main bronchus.

drainage and the lung examined is suctioned out by means of a catheter passed through the endotracheal tube. In a bilateral examination, the first lung injected is cleared of excess dye before proceeding with the opacification of the second lung. Following aspiration of the oral cavity, the endotracheal tube is removed after complete return of natural defence mechanisms, and oxygen is administered by mask. The patient is kept under close observation until fully awake. After a bronchographic procedure preceded by a bronchoscopic examination, the child is placed in a high humidity tent, if necessary, until all signs of laryngeal irritation have disappeared.

The radio-opaque medium which we use for bronchography is a chemically

stable, non-oily iodine preparation in an aqueous suspension known under the trade name of Dionosyl®. Its properties have been presented elsewhere[18] and need not be discussed at length here. Amongst its outstanding qualities, Dionosyl has no tendency to produce alveolar filling. Controlled respiration will not blow this contrast agent into the alveoli, provided it is used properly under conditions which will allow controlled minimum bronchial injection. This property is due to the fact that Dionosyl spreads evenly and adheres readily to bronchial mucosa. The lumen of the bronchi need not be flooded to procure uniform opacification. Thus a limited amount of dye is required and the resulting record presents a more natural appearance. Excretion does not involve the breakdown of Dionosyl to free iodine or inorganic iodide and for this reason it is said that Dionosyl is practically non-toxic. When used in minimum amounts, compatible with good results, Dionosyl is usually absorbed from the lungs within 48 hours.

Using the method described, we have performed to date a total of 40 bronchographies in infants and children under general anaesthesia. Unilateral bronchographies were performed on seven patients and bilateral procedures on 33 patients. The infant group included 14 cases ranging in age from two weeks to two years and an average age of 18.75 months. The child group included 26 patients ranging in age from three to nine years and an average age of 5.15 years. In the infant age group, the average quantity of Dionosyl used to opacify a single lung was 4.5 c.c. In the child age group, the average volume of Dionosyl required to opacify a single lung was 5.2 c.c.

Figures 4 and 5 illustrate technique and diagnostic results in a 14-day-old infant. Bronchography in this very young, transfused, erythroblastotic patient was indicated to rule out the possibility of bronchial agenesis or atresia on the side of a collapsed lung. Immediately following bronchography, a left endobronchial intubation was done under fluoroscopic control and a right thoracotomy was performed. Encysted empyema cavities were drained and the right lung was decorticated. The right consolidated atelectatic lung gradually re-expanded and the infant was discharged from the hospital in a good state of health one month after operation. This case illustrates that bronchographic procedures need not be withheld from the very young, so that they may benefit from life-saving surgical measures applied without undue delay.

Discussion

Although our method of general anaesthesia for bronchography in infants and children may appear to be complicated and to involve lengthy recommendations, the precautions herein set forth also apply to all methods of bronchography under general anaesthesia, however simple the technique may be. Indeed, oversimplification of technique, by reducing anaesthetic equipment to a minimum, deprives the patient of safety measures against a procedure which involves a serious risk. Whenever possible, a diagnostic procedure should not entail a greater risk to the patient than the subsequent therapeutic measure.

Although our anaesthetic apparatus is largely made up of standard anaesthetic

accessories, it could lead to a certain degree of carbon dioxide accumulation and anoxia in infants in the hands of the unwary. This would occur as a result of turbulence and rebreathing between the space separating the inspiratory valve on the Fink valve and the distal extremity of the Adams' connector. In spite of the oversize anaesthetic accessories, carbon dioxide accumulation and anoxia need not occur. The gases at the distal end of the apparatus are washed out and replaced periodically by opening the remote control clamp while simultaneously compressing the bag.

The entire apparatus could be made to handle more conveniently by incorporating plastic components throughout or by the use of lighter alloys. With the patient's head turned to one side, however, the apparatus need not be supported by the anaesthetist. It is made to rest conveniently on the X-ray table.

Since spontaneous breathing entails the opening of two inspiratory valves (one on the Ruben bag, the other on the Fink valve), respirations should always be controlled in infants and young children to avoid fatigue in the patients.

It is sometimes advisable, in bad risk cases involving extensive lung pathology, that the anaesthetic duties be shared by two anaesthetists. While one anaesthetist attends to parenteral requirements, the other gives his undivided attention to the inhalation procedure. In this way, less fumbling is likely to occur while working in the dark. Bronchography under general anaesthesia in older children and adults is performed with the same anaesthetic apparatus but here the special Metras catheters are used.

In the early stages of our experience with this method, we have witnessed the occurrence of spontaneous segmental atelectasis on two occasions during the fluoroscopic procedure. Presumably, these atelectatic areas were caused either by the injection of excessive amounts of Dionosyl with resultant segmental obstruction or by oversized injecting catheters which restricted alveolar ventilation. Smaller quantities of Dionosyl and greater precaution in the selection of injecting catheters of proper size have eliminated this complication.

In children, we have found that the Baxter plexitron administration set no. R33 very useful when used as a catheter to inject the Dionosyl. The needle adapter of the R33 is cut off and the Luer-type connecting tube at the other extremity is used for attaching the syringe containing the Dionosyl. Although somewhat soft, the tube may be guided fairly easily into either bronchus provided it is well lubricated with a water-soluble jelly. The smallest endotracheal tube that will accommodate this improvised catheter without causing serious mechanical obstruction is a no. 3 (Magill scale) plastic tube. For smaller endotracheal tubes, ranging in diameter from 0 to 2 (Magill scale), a no. 5 (Fr. scale) plastic infant feeding tube is used. Until a proper relative scale for endotracheal tube and corresponding catheter has been established, we recommend that the selection of injecting catheters be carried out extemporaneously on a trial and error basis before proceeding with the bronchography. A catheter which grossly obstructs the lumen of the endotracheal tube and interferes with ventilation, either spontaneous or controlled, should be rejected and replaced by another of smaller gauge.

Summary

A brief review of the principles involved in various methods of bronchography for infants and children under local and general anaesthesia is presented. The dangers involved and technical difficulties of performing bronchography without an endotracheal tube are discussed. Current methods of bronchography which include endotracheal intubation as part of the procedure are described and analysed.

The requirements for the performance of successful bronchography are stated. It is pointed out that oversimplification of anaesthetic technique exposes the patient to a dangerous hazard and leads to poor results. The use of explosive anaesthetic agents such as ether in the vicinity of an X-ray machine is considered a dangerous practice.

The radiation hazard during bronchography under general anaesthesia is discussed and a special anaesthetic apparatus with remote controls providing protection for the hands of the anaesthetist from the direct X-ray beam is described.

Details concerning the combined inhalation (non-rebreathing) and insufflation anaesthetic techniques involved are given. The principle of a common working airway shared by the anaesthetist and the endoscopist is adapted to the technique.

Recommendations pertaining to pre-anaesthetic preparation of the patient are given.

Induction of anaesthesia in the child is never attempted under duress. The danger involved in spraying the upper respiratory tract with a local anaesthetic agent in infants and children is stressed.

Maintenance of anaesthesia is accomplished with a non-explosive mixture of fluothane, nitrous oxide, and oxygen. Since coughing causes alveolar filling with the contrast agent, the patient is completely curarized to avoid a source of anoxia and obliteration of bronchial detail. Controlled respiration throughout the procedure is an essential part of the preliminary selective bronchographic technique described.

Dionosyl is the contrast medium selected. This dye produces a uniform coating and clings to the bronchial mucosa. There is no necessity to flood the bronchial lumen to obtain radiological results of good quality. When properly used, in limited amounts, controlled respiration will not cause Dionosyl to be blown into the alveoli.

Recommendations pertaining to the immediate post-anaesthetic care of the patient are given.

A report on a clinical study is included.

Acknowledgments

We are grateful to Mr. James Sharkay, Consultant Inhalation Therapist, for his valuable technical advice and to Mr. Gaston Royal, Technician, Engineering Department, Notre Dame Hospital, for his assistance in the design and construction of the remote control clamp herein described.*

*The remote-control clamp herein described is not commercially available.

RÉSUMÉ

Une revue succincte des principes qui se rattachent aux méthodes diverses de bronchographie sous anesthésie locale et générale chez les enfants est présentée. Les dangers et les difficultés d'ordre technique qui se présentent au cours de la bronchographie pratiquée sans intubation endotrachéale sont mentionnés. Les méthodes courantes préconisant l'usage d'un tube endotrachéal sont discutées.

Les conditions requisent s'appliquant à la conduite à tenir au cours de la bronchographie sont énoncées.

Le lecteur est mis en garde contre les techniques anesthésiques trop rudimentaires. Celles-ci peuvent être la cause déterminante non seulement d'un échec au point de vue résultat diagnostique mais, ce qui est plus sérieux, d'un accident grave chez l'enfant. L'utilisation de substances anesthésiques explosives, tel l'éther à la salle des rayons-x est une pratique dangereuse.

Il est essentiel de se prémunir contre les dangers d'irradiation au cours de la bronchographie sous anesthésie générale. C'est ce qui motive l'utilisation d'un appareil d'anesthésie à télécommande. Cet appareil apporte à l'anesthésiste un moyen de protection contre les faisceaux directs des rayons-x en lui permettant de tenir ses mains à l'écart.

La technique d'anesthésie préconisée associe l'inhalation et l'insufflation. Le principe d'une voie aérienne artificielle commune, partagée par l'endoscopiste et l'anesthésiste est adapté à la technique décrite.

Quelques conseils concernant la préparation pré-anesthésique du patient sont présentés.

L'induction de l'anesthésie chez l'enfant se fait toujours sans brusquerie. La vaporisation d'un agent anesthésique local au niveau des voies respiratoires supérieures est déconseillée, en raison du danger de réaction de surdosage.

La phase d'état de l'anesthésie est entretenue avec un mélange non explosif de fluothane, protoxide d'azote et oxygène. Puisque la toux contribue à l'inondation alvéolaire par l'agent de contrast, le patient est curarisé afin d'éviter non seulement le remplissage alvéolaire, source d'anoxie dangereuse, mais aussi, des résultats radiologiques inadéquats. La respiration est contrôlée pendant toute la durée de la procédure. Elle contribue à la technique de bronchographie sélective préliminaire décrite.

Le dionosyl® est la substance de contraste de choix. Cet agent semble avoir une affinité particulière pour la muqueuse bronchique et s'étale uniformément pour donner des résultats radiologiques satisfaisants, sans qu'il soit nécessaire d'inonder la lumière bronchique. Utilisé discrètement, tout en respectant les recommandations énoncées, le dionosyl® ne se propage pas jusqu'au alvéoles sous l'influence de la respiration contrôlée.

Les auteurs font quelques recommandations concernant les soins post-anesthésiques immédiats et font le relevé d'une étude clinique.

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THE ANAESTHETIST'S ROLE IN OPEN HEART SURGERY*

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ANAESTHESIA AS A SPECIALTY has taken great strides forward over the past fifteen years. This advancement has been due to the introduction of many new agents and techniques which have greatly improved surgical conditions and, therefore, increased the scope of both diagnostic and operative surgical procedures.

Recently, major developments have occurred which, once again, have opened up new surgical fields and have also caused the anaesthetist to re-explore the fields of pharmacology, physiology, and patho-physiology. In 1950, Bigelow and associates[1, 2] introduced hypothermia as a technique and since its introduction its use has become widespread in many fields of surgical endeavour. Hypothermia as a technique is usually handled by the anaesthetist, and has made it necessary for him to become an expert in the handling of such monitoring devices as direct pressure recordings, continuous electrocardiograms and electroencephalograms, blood pH and CO_2 determinations. In 1954, cross-circulation procedures, which were the fore-runners of total body perfusion in 1955, at the University of Minnesota[3, 4] made the team handling of surgical cases a necessity. Since 1955, many centres have developed "open heart" techniques and total body perfusion has become routine for the correction of intracardiac defects.

At the Vancouver General Hospital, open heart surgery was initiated in October, 1957, and since that time over two hundred cases have been operated on utilizing total body perfusion. These cases include children, infants, and adults with either congenital or acquired heart disease. The ages have ranged from eight months (10.5 lbs.) to 55 years. These cases have been selected entirely by the need for surgical intervention, with no particular regard to mortality figures. The over-all mortality has been about 25 per cent, a figure which compares favourably with other centres. The method of handling these cases was selected and developed in the animal laboratory‡ and it was found necessary to involve many individuals from various specialties before attempting clinical cases. These cases are now handled by a team which includes cardiologists, radiologists, surgeons, anaesthetists, haematologists, nurses from both operating room and recovery room, and technicians. Any surgical procedure with a mortality rate of 25 per cent would seem to require the undivided attention of all concerned. It is the role played by the anaesthetists on this team at the Vancouver General Hospital that I am principally interested in describing.

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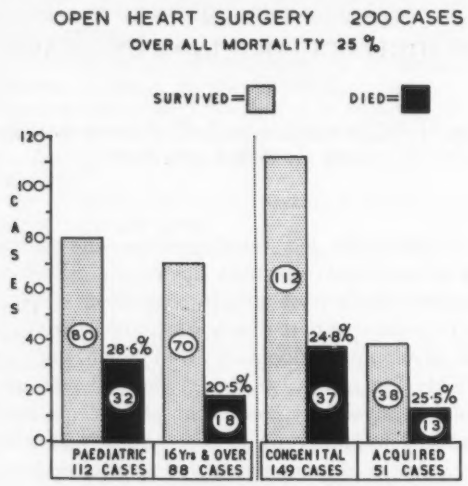

FIGURE 1. Mortality with reference to age and to congenital and acquired lesions. The mortality percentage is high due to the inclusion of the earlier cases.

The initial investigation of these patients is carried out by the cardiologist with the aid of the radiologist and haematologist. These investigations include history, examination, X-ray, electrocardiogram, right and/or left heart catheterization, angiocardiography, dye studies, and coagulation factors.

Before surgery is slated, the patient is presented at cardiac surgical rounds attended by all personnel concerned. Here the investigative findings are assessed and a diagnosis established. Once the diagnosis is established, the surgeons and anaesthetists must outline a plan to be followed in the operating room. This plan encompasses the management of perfusion, anaesthesia, surgery, and the postoperative care of the patient.

The perfusion is planned to suit the individual and his defect. Blood requirements, rate of perfusion, and estimated length of perfusion are considered. Also, special arrangements which may be necessary, such as coronary perfusion for aortic valvular surgery, method of cardiac arrest to be employed, and extra cannulations which may be used to decompress the left side of the heart, are discussed.

The anaesthesia is planned for the patient, taking into consideration such factors as age, cardiac output, pulse pressure, hypertrophy of one or both ventricles, arrhythmias, blood volume, the presence of cyanosis or myocarditis, pulmonary flow, and the hemodynamic status of the patient. Consideration is given to having the patient as free from cardiac failure as possible by use of digitalis, diuretics, and bed rest if necessary.

The management of the surgical procedure is discussed from the standpoint of position on the table, incision to be used, and the approach to the lesion. The method of repair is outlined. At this time, it is often necessary to entertain the

possibility of other lesions to be looked for at the time of surgery, as well as diagnostic pressure studies and saturations which may be of value during the procedure.

The postoperative care is outlined as to the necessity for respiratory assistance or tracheotomy or both. The postoperative cardiac management covers the control of blood volume, myocardial action, and arrhythmias. Consideration is given to the effect of hemodynamic changes occurring after the repair.

In the operating room, two anaesthetists are required: one concerns himself with the usual anaesthetic procedure, and the other with the supervision of the perfusion and the set-up and operation of the monitoring devices. The general duties of an anaesthetist are to maintain the patient during surgery. This involves pain relief, good surgical conditions, adequate oxygenation, maintenance of blood volume, and cardiovascular stability. Since the perfusion and the monitoring devices are necessary to maintain these patients during surgery, it is our opinion that the anaesthetist should control this equipment, and the surgeons, who are well aware of the limitations of the technique, are left free to handle the complex problems confronting them at the table.

The patient is premedicated lightly in order not to interfere with homeostatic mechanisms which, in the cardiac patient, are easily depressed. Some psychological preparation is usually necessary at this time, owing to the lack of sedative effect. The usual premedication is seco-barbital, grs. $\bar{i}$ to $\bar{i}\bar{s}\bar{s}$ for patients over ten years of age, 90 minutes preoperatively, meperidine, 0.75 mg. per lb. of body weight, and the usual dose of hyoscine, according to age, one hour preoperatively. These patients come to surgery awake, but relaxed and usually with no fall in arterial blood pressure. They are placed on a circulating blanket used for maintenance of body temperature, and given a "sleep dose" of thiopental sodium, slowly. Following an adequate dose of d-tubocurare and positive pressure ventilation with oxygen, the patient is intubated and respirations are controlled. Maintenance of anaesthesia is by means of nitrous oxide and oxygen 4:2 L. and the slow intraveous infusion of a .04 per cent meperidine solution. The total dose of meperidine ranges from 20 mg. to 150 mg., depending on the size of the patient and the length of the procedure. The predetermined dose of meperidine is run in at a rate calculated not to depress the blood pressure and to be absorbed as the chest is opened. Further d-tubocurare is usually necessary during opening of the chest, and just prior to perfusion. Following perfusion, nitrous oxide and oxygen only are required. The patient is usually awake and talking as the endotracheal tube is removed.

Recently, some patients with congenital heart disease have been intubated using succinylcholine and maintained on 0.5 to 1.0 per cent halothane. This concentration of halothane has not markedly reduced arterial pressures. However, without increased doses of curariform drugs, tidal volume was noticeably decreased, producing, in some cases, inadequate ventilaton. Also, since these patients did not respond more quickly at the end of the procedure, there did not seem to be any advantage to the use of halothane. We have thought it not advisable to use halothane in the presence of acquired heart disease with its myocarditis and the likelihood of myocardial depression. In fact, it has been our

opinion that the resuscitation of hearts from ventricular fibrillation or cardiac arrest has been more difficult in the presence of halothane anaesthesia. Owing to the electrical hazard in the operating room, no explosive agents are used. Careful observation of the patient and strict adherence to the fundamental principles of good anaesthesia is perhaps of more importance than the actual agents used.

Following induction, polyethylene catheters are inserted in the right and left antecubital veins and advanced well past the axilla. One of these is used for intravenous infusions and one to record venous pressures. A radial artery is cannulated to record arterial pressures; an alternative method may be used when the internal mammary artery is cannulated after the chest is opened. The electrocardiogram and electroencephalogram electrodes are inserted subcutaneously, a catheter is inserted into the bladder, a thermometer in the rectum, and the patient is positioned for surgery. Meanwhile, a technician has been assembling the pump oxygenator. The pump is of the finger type (Sigmamotor) and has a range of from less than 200 to 4,000 c.c. per min., which has been adequate for all perfusions. It is adjusted to operate in a totally occlusive manner. Hemolysis records have been kept on all cases, and show a range of increased plasma haemoglobin of from zero to 120 mg. per cent, with an average increase of 35 mg. per cent.

The oxygenator used is a bubble-type, as developed at the University of Minnesota.[4] If the calculated flow rate is over 1,500 c.c. per min., two oxygenators are used and the oxygen flow varies from 3 to 5 L. in each oxygenator. The bubbles are dispersed in debubbling chambers coated with a silicone preparation (Dow Corning Antifoam A). The oxygenated blood is collected in a helix reservoir, where further debubbling is accomplished as well as warming of the blood to body temperature by means of a thermostatically controlled water bath. The oxygenated blood is returned through the femoral artery, after passing through stainless steel filters (Abbott). The venous blood is collected in a venous reservoir by gravity drainage from the superior and inferior vena cavae. The pump is precalibrated to deliver flows of 50 c.c. per kilo per min. to adults and 60–70 c.c. per kilo per min. to children. Also, calibrations are carried out to enable the operator to increase the flow to a known quantity during perfusion if this be necessary to maintain adequate blood pressure. The various components, such as the reservoir and tubing of the oxygenator, are varied according to the size of the patient. Excessive pressure in the arterial line is kept to a minimum by inserting the largest possible catheter into the femoral artery. Intracardiac blood is gently suctioned off and returned for oxygenation. The reservoir is primed preoperatively with heparinized blood.

A careful calculation of blood loss is kept by weighing of sponges, and drapes and measurement of the amount suctioned. This loss is estimated throughout the case and losses are replaced intravenously with citrated blood. The gain or loss from the circulation during pumping is accurately measured after the perfusion and this figure is incorporated into the final estimations. Further checks on blood volume are made by pre and postoperative weighing of the patient, observation of the heart itself for venous filling following by-pass, as well as recorded arterial and venous pressures. One gram of calcium chloride is given intravenously for each litre of stored blood to counteract the citrate effect. During by-pass, the

lungs are inflated to a continuous pressure of 5–10 cm. of water with nitrous oxide and oxygen. The venous pressure, arterial pressure, electrocardiogram, and electroencephalogram are observed continuously as well as periodic arterial and venous oxygen saturations and pH estimations. By these observations, the anaesthetist can be assured that the patient is receiving adequate perfusion or, if not, steps can be taken to improve the perfusion. The perfusion is judged satisfactory if the arterial pressure is over sixty, the venous pressure is zero, the electroencephalogram after an initial period of slow increased amplitude waves, returns to a pre-perfusion tracing (usually one to two minutes), the arteriovenous oxygen difference is maintined, and the pH is above 7.4.

The surgeons are required to wait for one to two minutes for stabilization of the perfusion, and to ensure proper functioning of the cannulae before proceeding with the cardiotomy. The continuous observation of the electrocardiogram during perfusion (Lead II) has been most valuable. First, from the standpoint of avoiding heart block which usually occurs as a needle is placed through the conducting bundle; if this needle is removed without tying the suture, permanent damage may be avoided. In our series we have had two cases with permanent heart block which are surviving over one year, and one case which died post-operatively, not as a result of the block, as the heart rate was maintained by electrical stimulation of the myocardium. Secondly, the electrocardiogram is observed for signs of myocardial anoxia during intermittent aortic clamping or inadequate coronary perfusion during aortic valve surgery.

Before by-pass, the patient is heparinized with 1.5 mg. of heparin per kilo of body weight and, following perfusion, the heparin is neutralized with 3 mg. of protamine per kilo, or, more recently, hexadimethrine bromide (Polybrene-Abbott). Postoperatively, the blood-clotting mechanism is investigated for circulating heparin and extra protamine may be necessary. Cardiac arrest has been produced in the past, using potassium citrate, but this technique is now replaced by intermittent aortic clamping or, more recently, arrest produced by selective cooling of the myocardium by means of a heat exchanger and coronary perfusion.

In all cases, body temperature has been maintained by use of a warming blanket and warming the helix reservoir. It is possible, in the future, that total body cooling may be carried out during by-pass using the heat exchanger in order to reduce the rate of flow. However, with no real limitations in time or volume of our perfusion, this does not seem necessary.

After over two hundred cases of total body perfusion using Sigmamotor pumps and bubble oxygenator as described, we have found no serious limitations to the technique. The average length of by-pass has been forty-five minutes, with the longest being two hours and ten minutes, and the shortest, five and one-half minutes. There have been no serious complications directly related to hemolysis, inadequate oxygenation, silicone intoxication, or, we believe, to bubbles entering the circulation from the oxygenator. In our hands, the most serious disadvantages of this type of perfusion apparatus has been the time-consuming preparation of the tubing and the expense per case of the disposable parts (approximately $75).

In the post-anaesthetic room, the patient receives constant attention from all

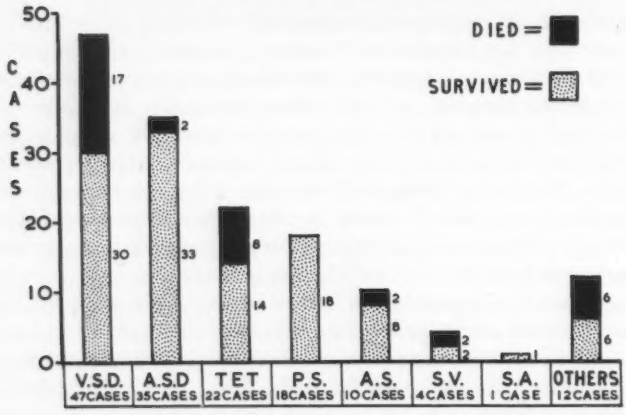

FIGURE 2. Mortality related to defect in congenital heart lesions.

personnel, until the respiratory exchange, arterial and venous pressures, cardiac rate and rhythm, and pH have stabilized. The respirations are assisted either continuously or intermittently using an intermittent positive pressure respirator (Bird). The cardiac action is observed for arrhythmias on a continuously recording cardioscope, pH determinations are made every fifteen minutes until stable, and acidosis is combatted by increasing respiratory excursion or the intravenous use of sodium bicarbonate. Blood is replaced as lost through chest drainage, and excessive bleeding is counteracted by coagulation factor estimations. Chest X-rays are taken routinely to check for haemothorax and atelectasis.

The post-operative care of these patients is very exacting and demands a thorough understanding of the defects involved and the surgical correction. Minute-to-minute changes in respiration, cardiac output, blood volume, myocardial irritability, and conduction mechanisms must be observed and combatted during the initial critical period. Any let down in the team effort at this stage may prove disastrous to a patient who, with careful scrutiny, would have recovered. Our mortality in these cases has been mainly due to diagnostic and surgical problems such as:

(*a*) failure to recognize additional defects such as a second ventricular septal defect or a ventricular septal defect associated with pulmonary stenosis or patent ductus arteriosus;

(*b*) pulmonary hypertension in the presence of ventricular septal defect, or ventricular septal defect associated with prolapsed aortic cusp;

(*c*) inability to correct the defect, as in advanced calcific deformity of aortic or mitral valves;

(*d*) Air emboli from the surgical field as in the repair of mitral valve disease, and emboli to the brain or to the mesenteric vessels from thrombi in the left atrial appendage;

(*e*) apparent inability of the myocardium to maintain adequate cardiac output postoperatively owing to myocarditis, ventriculotomy, or valvular deficiencies.

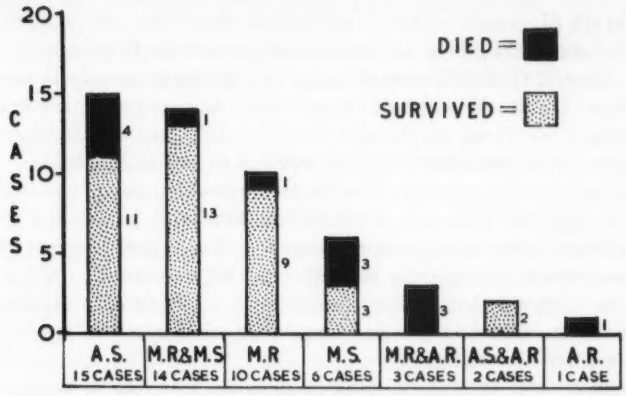

FIGURE 3. Mortality in acquired heart disease.

SUMMARY

There are many types of pumps and oxygenators in use today, all of which, when handled properly, are satisfactory. No doubt, in the future, there will be many improvements in both pumps and oxygenators. However, at this time, the limitations of open heart surgery do not lie in perfusion techniques, but rather in accurate diagnosis of the defect or defects and the surgical ability to correct these defects. The anaesthetist may broaden the scope of surgical correction in these cases by taking his proper place on the open heart team and maintaining the patient during the procedure, leaving to the surgeon only those problems involved in correction of the defect. In order to do this, the anaesthetist, besides his usual anaesthetic duties, must take an active interest in the diagnosis and have a real understanding of the complexities of the defect to be repaired, as well as a thorough understanding of perfusion problems and the postoperative complications.

RÉSUMÉ

Au cours des cinq dernières années, la chirurgie à coeur ouvert ou la chirurgie cardiaque où l'on emploie une pompe et un oxygénateur s'est développée rapidement. Dans les endroits où l'on a obtenu du succès dans ce domaine, on a attaché une grande importance à l'équipe dirigeant les opérations. Nous prenons l'opportunité actuelle pour préciser le rôle de l'anesthésiste dans cette équipe, maintenant que la période d'adaptation est passée. Au début, on attachait une grande importance aux méthodes employées pour dévier la circulation mais actuellement, depuis l'apparition de plusieurs types d'oxygénateurs et de pompes qui se sont avérés à point, on a concentré l'intérêt sur le diagnostic, les soins au cours de l'opération, la correction chirurgicale et les soins post-opératoires.

Après avoir opéré maintenant au-delà de 200 cas de lésions acquises ou congénitales chez des enfants ou chez des adultes, les anesthésistes ont assumé et joué un rôle de première importance comme membres de cette équipe depuis

la précision du diagnostic—qui est un facteur important au point de vue de la conduite de l'anesthésie, de la méthode de perfusion et de la technique chirurgicale proposée—jusqu'à la salle d'opération où l'anesthésiste assume la responsabilité de l'anesthésie, la mise en marche de la perfusion, le fonctionnement des moniteurs qui renseignent sur l'état du malade. Voilà ce qui laisse au chirurgien l'entière liberté d'essayer de résoudre les problèmes qui se présentent à lui.

Parmi les moniteurs, on compte: un électrocardiogramme, un électro-encéphalogramme, un appareil mesurant la pression artérielle et la pression veineuse, un oxymètre mesurant les saturations en oxygène et des appareils mesurant l'activité métabolique. Après une grande série de cas, nous estimons qu'il s'impose de continuer ces études pour mieux comprendre les réactions du malade au cours de ces opérations et aussi à cause du changement continuel des méthodes de perfusion et de correction chirurgicale.

L'anesthésiste suit la malade dans la salle de réveil et se concentre sur les problèmes de la respiration et du métabolisme et fait équipe avec le chirurgien pour rétablir l'équilibre chez le malade.

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HALOTHANE (FLUOTHANE) ANAESTHESIA FOR PAEDIATRIC CARDIAC SURGERY

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HALOTHANE, 1,1,1,-trifluoro-2,2, bromochloroethane, is a volatile halogenated hydrocarbon, which was introduced in 1956 as a general anaesthetic agent.[1, 2, 3] It appeared during an era of enlightenment in anaesthesiology, and more zeal has been directed to the investigation and clinical application of halothane than immediately followed the introduction of any other anaesthetic agent. In the past few years, halothane has established itself as a valuable addition to the armamentarium of anaesthetic drugs.

There has been an increasing tendency in recent years to utilize electric devices during cardiac surgery. The surgeon may require electrocautery or the electric saw; the pumps are electrically operated, and the anaesthesiologist has adapted electronic instruments to aid him in the care of the patient during the operation. Frequently, therefore, a non-explosive anaesthetic technique is mandatory. Prior to the introduction of halothane, there was no non-inflammable inhalation anaesthetic which was suitable for administration to patients during heart surgery. Halothane, however, possessed certain features which merited consideration. Since early studies of halothane emphasized the myocardial depressant properties of the new drug,[1, 4] there was an initial reluctance to administer halothane to patients with myocardial disease.

When halothane was first employed at Indiana University Medical Center, it was observed that patients with acquired and congenital heart disease tolerated the agent very well, if light levels of anaesthesia were maintained. There appeared to be a clearly defined need, at that time, for a non-inflammable inhalation anaesthetic which could be used during cardiac surgery. It was decided, therefore, to evaluate halothane anaesthesia in this role. The early results were encouraging and the study was continued.

The following report describes the clinical experiences with the administration of halothane anesthesia to 200 children during the surgical correction of congenital cardiac effects.

CLINICAL MATERIAL

The data which is reported has been obtained from medical records at Indiana University Medical Center. All of these patients were less than 16 years of age (Fig. 1). A variety of cardiac lesions and operations were encountered during this series (Table I). Extracorporeal circulation and moderate hypothermia were

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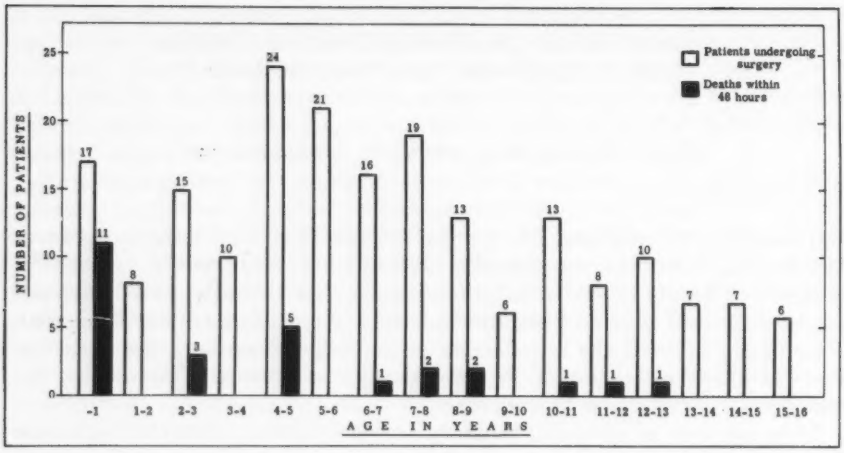

FIGURE 1. The age distribution of the 200 children, and of those children who died within 48 hours.

used during 108 of these operations to allow the defects to be repaired under direct vision, but were not required in the remaining operations.

CLINICAL METHOD

The children received 0.1 mg. of morphine sulfate or 1.0 mg. of meperidine hydrochloride (Demerol®) per pound of body weight, by intramuscular injection, one and one-half hours before surgery. Ten mg. of morphine or 100 mg. of meperidine were the maximal amounts administered to any patient.

The first seventy of these patients who underwent open heart surgery received neither atropine sulfate nor scopolamine hydrobromide. The anticholinergic drugs were omitted at the behest of the surgeon since it was feared that they might antagonize the effect of acetylcholine, which was employed when necessary to produce elective cardiac arrest during the cardiotomy. Atropine was injected intravenously during the course of some of these operations, and later, preoperatively, by intramuscular injection. Elective cardiac arrest could still be produced in these patients by the injection of acetylcholine. The remaining patients received atropine or scopolamine by intramuscular injection, in doses appropriate for their age and weight, when the narcotic was injected.

In the early part of this study, anaesthesia was induced by the inhalation of 50 per cent nitrous oxide in oxygen, to which halothane was added gradually from an accurately calibrated vaporizer. This technique allowed a rapid and quiet induction of anaesthesia, and the endotracheal intubation. Two serious difficulties were encountered. High concentrations of halothane, up to 2.4 per cent, were used to achieve these results, and some of the children developed alarming hypotension and bradycardia in response to these concentrations. It was possible, fortunately, to resuscitate all of these children. The other difficulty arose

TABLE I

THE DISTRIBUTION OF THE 200 CHILDREN, AND THOSE CHILDREN WHO DIED WITHIN 48 HOURS, IN RELATION TO THE TYPE OF CARDIAC DEFECT

	Number of patients	Number of deaths within 48 hours
Intracardiace (open heart) surgery		
Atrial septal defect	33	1
Ventricular septal defect	23	4
Tetralogy of fallot	18	7
Pulmonary stenosis	13	2
Anomalous pulmonary venous return	8	2
Miscellaneous	13	2
TOTAL	108	18
Extracardiac surgery		
Transection of patent ductus arteriosus	36	0
Blalock or potts procedure	24	1
Pulmonary artery banding	12	6
Pulmonary valvulotomy	10	0
Resection of coarctation of the aorta	8	0
Creation of interatrial septal defect	2	2
TOTAL	92	9

when there was some delay in the introduction of the endotracheal tube into the trachea. Laryngospasm or severe coughing occurred, and the accompanying hypoxia was a most disturbing and undesirable feature. The use of this technique, therefore, to induce anaesthesia in these patients was abandoned.

Following this decision, anaesthesia was induced, when the child permitted venepuncture, by the slow intravenous injection of a 1.0 per cent solution of methohexital sodium (Brevital®).[5] Oxygen was then administered by intermittent positive pressure, and succinylcholine solution was injected intravenously to provide muscular relaxation, so that an endotracheal tube could be inserted into the trachea. With the other children, the induction of anaesthesia was accomplished by the administration of divinyl ether (Vinethene) and diethyl ether by the open drop method, and endotracheal intubation was performed when the muscular relaxation appeared adequate.

During the operation, a steady level of anaesthesia was maintained by the administration of a mixture of 50 per cent nitrous oxide in oxygen, to which halothane was added in concentrations up to 0.8 per cent. Adjustments were made within this range to provide immobility of the patient. Electroencephalograms demonstrated patterns 1 or 2 when these concentrations of halothane were employed.[6] The halothane was administered in oxygen, instead of nitrous oxide and oxygen, when warranted by the patient's condition. A flow rate of eight litres of anaesthetic mixture per minute and a semi-closed to and fro absorption system allowed the elimination of endogenous carbon dioxide. Respiration was controlled throughout the operation. Apnoea was produced quite readily, in most of the patients, by hyperventilating them with the anaesthetic

mixture. Succinylcholine was administered, when indicated, to reinforce the effect of the hyperventilation, and to ensure the avoidance of coughing at critical points in the surgical procedure. The succinylcholine was injected as a 0.2 per cent solution by continuous intravenous drip infusion with children over two years of age. With the younger children the muscle relaxant was injected intermittently, in doses of 1.0–1.5 mg. per pound of body weight by the intramuscular or intravenous routes to avoid the danger of infusing excessive amounts of fluid.

Halothane was not administered to the patients during the cardiopulmonary by-pass in open heart operations. Previous experience had shown that the dangerous cardiac arrhythmias occurred almost exclusively during or following the cardiotomy. When halothane was first introduced it had the reputation of enhancing myocardial irritability.[7] It appeared advisable, therefore, to refrain from exhibiting halothane during this stage of the operation. Instead, methohexital sodium (Brevital®) was introduced into the oxygenator chamber to provide sedation, and succinylcholine solution was injected into a peripheral vein to ensure complete immobility of the diaphragm.

Controlled respiration was maintained until surgery had been completed, when oxygen was administered through the endotracheal tube and a complete tracheobronchial toilet was performed. The endotracheal tube was removed when the child appeared to be awakening, and the administration of oxygen was continued with a face mask. When spontaneous ventilation appeared to be adequate, the patient was taken to the recovery room.

Results

The number of deaths which occurred during surgery or during the first 48-hour postoperative period are shown in relation to the cardiac defect (Table I) and to the age of the patient (Fig. 1). It was the combined opinion of the anaesthesia and surgical teams that the anaesthetic agents were not responsible for any of these deaths. Mortality figures in cardiac surgery are misleading unless special regard is paid to the physical condition of the child and to the nature of the surgery. The table includes children with such severe cardiac disease that death was inevitable in the immediate future, and surgery was offered in desperation as the only hope for amelioration. It was the rare success which justified the acceptance of these moribund patients. If the operation failed to improve their condition appreciably, they succumbed to the stress of the operation during or immediately following the procedure.

Anaesthesia was induced in fifteen children by the inhalation of a halothane, nitrous oxide, and oxygen mixture. Nine of these children developed severe hypotension and bradycardia. An emergency thoracotomy was necessary in only one patient, and the heart was still beating when it was exposed. All of these patients responded to the elimination of the halothane, to ventilation with oxygen, and to the intravenous injection of atropine sulfate. The operation was abandoned in the case of the child who had the thoracotomy, but was continued with the other children using light halothane anaesthesia. No further difficulties were encountered during these operations which could be attributed to the use of halothane.

The systolic blood pressure was depressed 10–20 mm. Hg and the pulse rate 20–30 beats per min. from the pre-anaesthetic resting level in most of these children. With patients who were undergoing open heart surgery, it was found that if some degree of hypotension was present the intramuscular injection of metaraminol (Aramine®) prior to the cardiopulmonary by-pass allowed a higher pressure to be maintained during the perfusion. Severe hypotension did occur during some of these cases, but was associated with rapid blood loss, surgical manipulations within the thorax, and the terminal deterioration of the moribund patients.

Manipulation of the heart to allow the corrective surgical procedure is almost invariably accompanied by the appearance of various types of cardiac arrhythmias, whichever anaesthetic agent has been selected. There were other arrhythmias which developed during anaesthesia. Ventricular extrasystoles occurred with 25 patients, auricular extrasystoles with 15 patients, and nodal rhythm with eight patients. These were transitory and appeared to be associated with inadequate ventilation or surgical manipulations. Two patients demonstrated nodal tachycardia and one patient ventricular tachycardia during open heart surgery. They resumed a sinus rhythm within 24 hours.

Ventricular fibrillation was observed with 11 patients. This occurred during the cardiopulmonary by-pass in 10 children, who were undergoing open heart surgery. Acetylcholine had been injected to produce elective cardiac arrest in eight of these patients. This complication has been noted following the use of acetylcholine and potassium citrate for this purpose.[8] It was difficult to implicate halothane in any of these cases, since none had been administered during the by-pass. Any residuum from the preceding stage of the operation can be assumed to have been eliminated in the oxygenator chamber. It was possible to revert the ventricular fibrillation to a sinus rhythm in nine of these patients.

Cardiac asystole occurred during operations on six children. All of these children were moribund before anaesthesia was commenced. There was no reason to believe that they would have survived if some other anaesthetic technique had been selected.

Samples of arterial blood were obtained from 23 children during cardiac surgery (Fig. 2). The first sample was obtained when the patient was breathing spontaneously following the endotracheal intubation, and the effects of the drugs which had been employed to induce anaesthesia had abated. Mild respiratory depression was evident. The second and third were removed during the operation and demonstrated that adequate arterial carbon dioxide tensions could be maintained with adequate ventilation of the patient. The final sample was obtained just before the child was removed from the operating room. The patients were awake and were breathing spontaneously. The near normal tensions of carbon dioxide in the arterial blood confirmed the fact that adequate ventilation returned rapidly following anaesthesia with low concentrations of halothane.

Discussion

The skill and experience of the anaesthesiologist are the most important factors in the successful conduct of anaesthesia during cardiac surgery. The significance

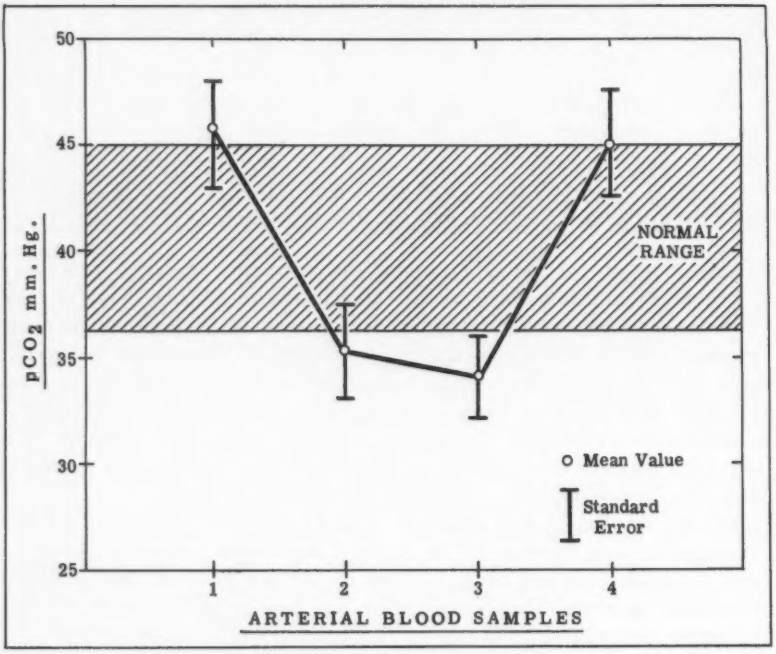

FIGURE 2. A graphic representation of the tensions of carbon dioxide in the arterial blood of 23 children, during and immediately following surgery, in relation to the normal range.[14]

of the selection of anaesthetic agents, however, should not be belittled. Although most of the popular anaesthetic agents have been used successfully to anaesthetize these patients, there appeared to be a need for a satisfactory non-inflammable inhalation anaesthetic so that electric instruments could be used safely during the operation. Halothane satisfied this requirement, and this study has shown that it was eminently suitable to provide anaesthesia during cardiac surgery, when administered in low concentrations.

Light halothane anaesthesia provided amnesia, analgesia, and immobility—the basic requirements of general anaesthesia during this type of surgery. A steady and predictable level of anaesthesia was maintained throughout the operation with delightful simplicity by the adjustment of a calibrated vaporizer. The absence of significant respiratory depression in the immediate postoperative period was demonstrated by the measurement of the tension of carbon dioxide in arterial blood. Halothane, when used in low concentrations, was rapidly eliminated following the termination of anaesthesia. This allowed a rapid awakening of the patient and a rapid return of adequate ventilatory exchange. Apnoea was obtained readily in most of these children when they were hyperventilated with the anaesthetic mixture. Muscle relaxants, therefore, were employed merely to reinforce this effect, thus reducing the amount of the relaxant which was

required during the operation. This factor also favoured the rapid return of adequate ventilation following the termination of anaesthesia.

The ability to administer anaesthetic mixtures containing high concentrations of oxygen is essential when anaesthetizing patients with cardiac disease. Any desired concentration of oxygen could be offered to these patients without any appreciable alteration in the concentration of halothane.

The most strenuous arguments against the employment of halothane when the patient has cardiac disease have been based on the alleged myocardial depressant effect of the drug. Investigations in humans and animals, however, have clearly demonstrated that there is no appreciable cardiovascular depression during light halothane anaesthesia.[9] Measurement of the cardiac output and the ventricular contractile force have revealed no significant decrease in these parameters when low concentrations were used.[10, 11] In this clinical study, light halothane anaesthesia provided adequate anaesthesia to patients with heart disease throughout the operation. When the concentrations were maintained at 0.8 per cent or less, no cardiovascular depression of any consequence occurred which could be attributed to the action of halothane.

There appears to be little doubt that, in common with other anaesthetic agents, deep halothane anaesthesia causes depression of cardiac function. Severinghaus and Cullen found a decrease in the cardiac output and the stroke volume of human volunteers who were artificially ventilated with 1.5 per cent halothane.[4] They concluded that this was the result of myocardial depression. McGregor and his colleagues[10] showed that the cardiac output was decreased in normal children during moderate or deep anaesthesia. They were reluctant to ascribe this entirely to myocardial depression. Sprouse, Thrower, and their group[11, 12] measured the ventricular contractile force and the cardiac output in humans and animals during deep halothane anaesthesia, and found a decrease of 40–50 per cent in each from the control values. They noted a return in the cardiac output to near control values when atropine was injected, and concluded that the decrease was associated with the bradycardia, rather than with myocardial depression. Cardiac depression was observed in the patients with cardiac disease, included in this series, when anaesthesia was induced with high concentrations of halothane. Alarming hypotension and bradycardia appeared abruptly, and there was little doubt amongst those present that cardiac arrest would have been the obvious sequel if resuscitation had not been carried out promptly. A thoracotomy was performed on one of these children who had received 2.4 per cent halothane, and although the heart was still beating and appeared well-oxygenated, there could be little question that there was dangerous depression of cardiac function. It is true that not all of the children in whom anaesthesia was induced with high concentrations of halothane demonstrated this severe depression, but the frequency of this dangerous complication was sufficient to condemn this technique.

There are no non-inflammable inhalation anaesthetic agents except halothane and nitrous oxide which are suitable for anaesthetizing patients during cardiac surgery. Other non-explosive anaesthetic techniques depend on the supplementation of nitrous oxide and oxygen anaesthesia by the intravenous injection of

bartiturates and narcotics. Inhalation anaesthetics can be eliminated readily through the respiratory tract by ventilating the patient. Drugs which have been injected intravenously must await complex pharmacophysiologic processes, which may be retarded when the patient has been subjected to the rigours of cardiac surgery. The facility with which inhalation agents can be removed from the patient allows more rapid correction of the effects of an overdose, greater ease in maintaining a steady level of anaesthesia, and a greater assurance that hypnosis and respiratory depression will be minimal when anaesthesia has been terminated.

Halothane anaesthesia has been used exclusively at this institution during the past three years with patients undergoing open heart surgery. Any comparison of these patients with those who underwent intracardiac surgery prior to the use of halothane is not valid. The decrease in the frequency of anaesthetic and surgical complications, and in the mortality rate, is due primarily to the increase in the skill of the surgical and anaesthesia teams. There are certain heart operations, however, which have become standardized with regard to surgical technique and anticipated complications, so that a comparison of the effects of the different anaesthetic agents employed appears to be reasonable. Two groups of 68 children were compared. The first group received halothane anaesthesia, and the second cyclopropane or ether anaesthesia. They underwent surgery to correct either a patent ductus arteriosus, a coarctation of the aorta, or, by extracardiac anastomosis, a tetralogy of Fallot. One patient in each group died during the postoperative period. Cardiovascular depression and cardiac arrhythmias occurred with nearly equal frequency in both groups. These appeared to be due to surgical manipulations or errors in anaesthetic technique, and not to the anaesthetic agents. The arterial blood pressure was usually maintained at a higher level during cyclopropane anaesthesia, and the pulse rate stabilized at a lower rate during cyclopropane and halothane anaesthesia. No patients in either group manifested severe hypertension during the postoperative period following the surgical repair of coarctation of the aorta.[13] There did not appear to be any significant difference in the response of the patient during anaesthesia, whichever anaesthetic agent was employed.

Summary and Conclusion

Light halothane anaesthesia was administered to 200 children during cardiac surgery. The principle advantages of halothane, when compared to other inhalation anaesthetics, was its lack of flammability. The children tolerated the agent very well if low concentrations were employed, but frequently developed alarming signs of cardiac depression with high concentrations of halothane.

Halothane could not be recommended to induce anaesthesia for cardiac surgery. It was safe, and provided satisfactory anaesthesia, when light halothane anaesthesia was maintained throughout the operation. Light halothane anaesthesia merits serious consideration when a non-inflammable anaesthetic technique is required during cardiac surgery.

RÉSUMÉ

L'apparition, au cours des dernières années, d'appareils électriques comme adjuvants au cours de la chirurgie cardiaque, nous a placés dans l'obligation d'employer au cours de cette chirurgie un agent anesthésique non explosif. Quand l'halothane est apparu comme agent anesthésique, on a insisté sur son effet dépresseur sur le myocarde. Toutefois, l'expérience révèle que, si l'on emploie de faibles concentrations, même les porteurs de maladies cardiovasculaires le tolèrent très bien.

Au cours de ce travail nous décrivons, chez 200 enfants ayant subi de la chirurgie cardiaque, comment nous avons administré l'halothane avec le protoxyde et l'oxygène. Nous avons observé que, sinous employions de fortes concentrations d'halothane, les enfants présentaient souvent des dépressions cardiovasculaires inquiétantes. Pour l'induction, nous avons donc employé d'autres agents anesthésiques. Pour le maintien de l'anesthésie, l'halothane, administré à une concentration ne dépassant pas 0.8% avec du protoxyde et de l'oxygène, a produit un niveau d'anesthésie tout-à-fait convenable.

Nous ne pouvons pas attribué à l'halothane aucune des morts survenues au cours de la chirirgie et au cours des quarante-huit heures qui l'ont suivie. Nous n'avons recontré aucune difficulté importante aussi longtemps que nous n'avons pas dépassé une concentration de 0.8%. La recherche de la tension du gaz carbonique dans le sang artériel nous a démontré que toute dépression respiratoire attribuable à l'halothane disparaissait rapidement à la fin de l'anesthésie.

Nous pouvons conseiller l'usage de l'halothane au cours de la chirurgie cardiaque lorsqu'il s'impose d'employer un agent non explosif.

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POSTOPERATIVE ADMINISTRATION OF METHYLPHENIDATE

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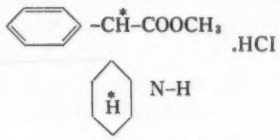
THE EXTENSIVE RANGE of drugs known as stimulants can give rise to some confusion of judgment as to their ultimate usefulness. However, classification according to their pharmacodynamic properties will serve as a guide to their appropriate application. Among the central stimulants there are several groups, and the drug under discussion in this paper is associated with the phenyl-piperidyl-acetic acid derivatives. Methylphenidate or Ritalin® is phenyl piperidyl[2] acetic acid methylester. This drug has been shown to have a particularly strong central stimulating action in both humans and animals.



According to the observations on animals by Gross[1] and co-workers, psychomotor stimulation is produced by methylphenidate in the unanaesthetized animal. The intensity of activity produced by a dose of 0.5 to 1.5 mg./kilo varies from mild restlessness to complete exhaustion. Larger doses of 2 mg./kilo and above can produce compulsive movements of such great intensity that the animals run around until they reach a severe state of exhaustion with subsequent death. Some typical individual movements observed in dogs were twitching and turning of the head associated with movements of the eyelids and ears. An acceleration of respiratory rate was noted by Gross[1] after an intravenous injection of 1 mg./kilo, and apparently with the higher range of doses this was increased. A distinct analeptic effect was found to be produced by methylphenidate after the administration of thiopentone. Doses of 25 mg./kilo of the former administered subcutaneously completely abolished the thiopentone effect. This arousal persisted up to the fourth hour after the injection of the methylphenidate, and was accompanied by a considerable increase in motility. However, the analeptic effect of this drug was only slight on the longer acting barbiturates. The respiratory stimulation in the anaesthetized animals was even more marked than in the unanaesthetised. The cardiovascular changes were a prolonged rise in blood pressure and an acceleration in heart rate.

A clinical survey was conducted on patients during the immediate postoperative period, that is, in the recovery room. A double blind method of administration was employed, the methylphenidate or placebo being supplied by the hospital

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pharmacy. Minor operation cases only were included in the trial, so that a fairly constant range might be observed. On arrival in the recovery room the blood pressure was checked, and immediately afterwards a spirometry recording was made. Following the recording, 1 c.c. of the solution supplied was injected intramuscularly, and a careful watch made for the first sign of arousal of the patient. Fifteen minutes after the injection, a second spirometer recording was made, regardless of whether the patient was awake or not. In all, 294 patients were observed, and records from the pharmacy eventually revealed that 174 patients had received Ritalin®, and 120 the placebo. If the solution administered was methylphenidate, 1 c.c. contained 20 mg. of the drug.

Results

Certain variables entered into the investigation and these will be considered before the analysis of the results is attempted.

(1) Although the preoperative medication was kept fairly constant by the administration of meperidine and atropine, a small number of patients received anti-emetics with the sedation. On examining the distribution of the additional drugs in the two groups, there appears to be no statistically significant difference in the numbers.

(2) Similarly, the anaesthesia was slightly variable, but out of the total of 294 patients, 265 received thiopentone combined with nitrous oxide and oxygen.

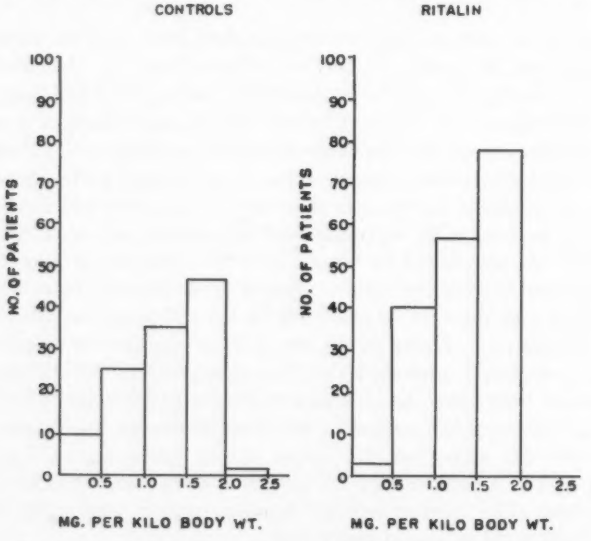

Figure 1. The amount of meperidine administered has been worked out in mg./kilo body wt. The distribution is moderately asymmetrical, and the values when tested by the statistic of χ^2 show that P is 0.20, indicating that there is no significant difference in distribution of dosage in the two groups of patients.

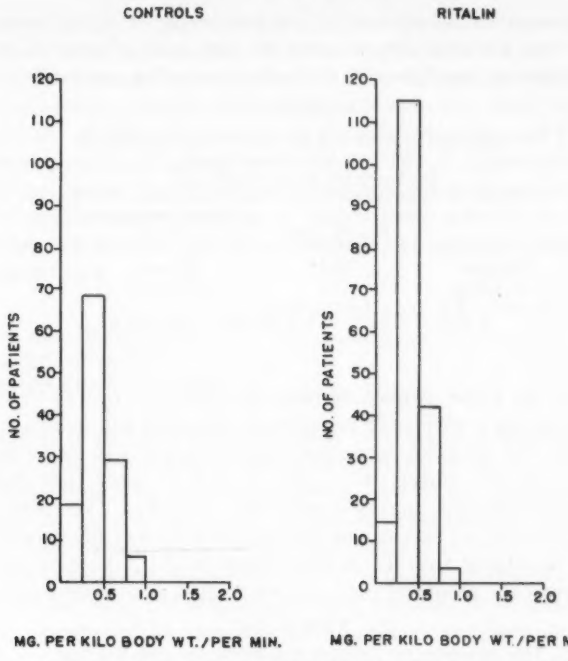

FIGURE 2. The amount of thiopentone given to each patient has been presented in mg./kilo/min. (per min. of actual anaesthesia). This histogram, too, shows moderate asymmetry and the value of P is 0.3, which is not significant.

The remaining 29 were given cyclopropane, trichlorethylene or halothane in addition. This distribution was examined as in the case of preoperative sedation, and here also there was no significant difference in the distribution.

There is some disagreement as to the specific antagonistic influence of methylphenidate on the narcotic action of meperidine, and so it was decided to consider the frequency distribution of this drug, as well as that of the thiopentone. These distributions are shown in the form of histograms (Figs. 1 and 2).

After having considered these additional factors and presuming that the drugs and the amount administered are fairly evenly distributed in the two groups of patients, the main results can be analysed. There were four major points of consideration: the time taken for the patient to recover from the anaesthetic; the alteration in minute volume of respiration after the injection given in the recovery room; the changes in blood pressure in response to the injection; the incidence and type of side-effects.

There was a strong tendency to believe that patients who received the methylphenidate were more alert on the return to consciousness, according to observations recorded at the time when the nature of the injection was not known. However, an analysis of the psychic function is too difficult for one not trained

in psychoanalysis, and consequently I am not prepared to say more than that this alertness was a visual appreciation on the part of the observers. More detailed attention has been given to the variation in the awakening time.

TABLE I

A Statistical Analysis of the Recovery Time in the Two Groups of Cases
(P. being 0.5 is not significant)

	No. of cases	Mean awakening time
Ritalin	162	23 mins.
Controls	113	26 mins.
S.E.D. = 5.5217	t = 0.5415	P = 0 5

Table I shows the mean awakening time after the injection in the two groups. All patients awakening within 10 minutes of receiving the injection have been eliminated from this analysis, since one cannot expect any real effect from an intramuscular injection within that time. It will be seen that the difference between the two arithmetic means is only three minutes. The standard error of the difference between the means is so near to the values of the means themselves that it is not surprising that P. is .50. Consequently, there is no significant difference between the recovery times in the treated and control groups.

TABLE II

The Significant Difference between the Values of the Minute Volume in the Methylphenidate and Control Groups
(P = .01)

	No. of cases	Mean gain
Ritalin	174	857 ml.
Controls	120	517 ml.
S.E.D. = 118.01	t = 2.881	P = .01

Table II presents the results of analysis of the spirometry recordings made while the patient was in the recovery room. The difference between the mean increase in each group is shown, and it is interesting to note that there is a definite increase in the mean minute volume in the methylphenidate group, and the statistical analysis gives the value of P. as 0.01, that is, a significant value. This increase in minute volume was produced more by an improved tidal air measurement rather than by an increase in respiratory rate. That this was owing to general arousal was not the case, since patients who continued to sleep on for anything up to 30 minutes after the second spirometry recording sometimes showed this improved respiratory activity.

The blood pressure changes were slight and the difference between the two means of the systolic readings was 5 mm. Hg. This cannot be presented as a reliable figure, since a number of people were concerned in the recording of these pressures, and consequently a certain degree of personal error would enter into

the evaluation. One might presume from such a small difference that methylphenidate did not have any marked effect on the blood pressure.

The side-effects produced by any drug are of considerable importance, since one which can produce a state of wild excitement is hardly likely to recommend itself to clinical use. Previous investigators, Gale,[2] Percheson, and Carroll,[3] have recorded marked psychical disturbances following the administration of methylphenidate. In this series, there were nine patients who showed signs of overstimulation of psychomotor function, of which there were six in the methylphenidate group and three in the control series. The degree of disturbance can be classified as follows:

	Methylphenidate Group	*Control Group*
General restlessness	3	1
Crying and restlessness	2	2
Crying and thrashing around	1	0

Since some of control patients showed signs of reaction, it is felt that there must have been some underlying mental disturbance at the time. It is interesting to note that the constant head movement seen in animal experiments was noticed in some of these patients. Fortunately the period of restlessness did not interfere with the spirometry recordings since all but two had not shown signs of arousal at the time of their second recording. The two patients who were awake had settled down by the time the second recording was due to be made. Two very restless patients could not be recorded, but they have not been included in this series.

A very limited experience of the use of methylphenidate on the anoxic newborn infant indicated that the drug had definite stimulant effect, especially in the presence of sedation and general anaesthesia. Two infants born by caesarean section and one by a difficult forceps delivery were given methylphenidate, 2 mg. administered intramuscularly after the usual methods of resuscitation (including intubation) had been applied without success. In each case it was approximately five minutes after birth that the methylphenidate was injected. In all three cases there was a definite response within 10 minutes, although in the case of the forceps delivery, the infant showed marked cerebral irritation for at least 24 hours after. Since the infant weighed 9 lb. 6 oz., the dose of methylphenidate was not thought to be excessive, and it was presumed that the cause of crying and increased tone might have been due more to the difficult delivery than the stimulant. The infant improved with sedation.

Discussion

It has been stated that an analeptic with a brief effect (even though the intensity of effect is great) will abolish the anaesthesia only temporarily when it is due to a drug with a prolonged action. According to Gros *et al.*[1] the analeptic effect of methylphenidate is only slight when tested against large doses of barbiturates, with the exception of thiopentone, and is not comparable with that of true analeptics, although its ability to produce psychic stimulation has caused

it to be utilised for barbiturate depression (Carter and Maley,[4] Gale,[2] and Smith and Adriani[5]). Plummer and Yonkman[9] point out that the effect of methylphenidate is in contradistinction to that of tranquilizers, since its main pharmacological action is the stimulation of the mesencephalic reticular formations and accentuation of the arousal mechanism. The effect of methylphenidate on the patients in this survey does not produce significant results in arousal time, with a dose varying between 0.2 to 0.5 mg. per kilo body weight (the majority of patients receiving 0.3 or 0.4 mg. per kilo). Gale[2] noted that doses above 0.1 to 0.2 mg./lb. were effective in shortening the recovery time, but that larger amounts were probably ineffective. Yet, Dokin[6] records a significant reduction in the recovery time when methyl-phenidyl-acetate (1 mg./kilo) was added to thiopentone (25 mg./kilo) in a controlled series of dogs. It may be that the patients in this series were not sufficiently depressed by the anaesthesia to show any significant improvement in awakening time over the control cases. This conclusion would concur with Gale's[2] statement that the effectiveness of methylphenidate seems to be related more to depth of depression than the actual depressant agent. Methylphenidate does not appear to be antagonistic to one group of drugs only, as in the case of levallorphan, since a number of investigators (Ferguson *et al.*[7] and Carter *et al.*[4]) have reported beneficial results of its use in central nervous system depression caused by chloroform, reserpine, barbiturates, and general anaesthesia.

The stimulant effect on respiration was significant in this experiment, but it is interesting to note that there did not appear to be any relationship between the dose of thiopentone and the degree of respiratory stimulation. Gale[2] pointed out the lack of correlation between the optimal dose and the amount of thiopentone administered, and concluded that methylphenidate acts not as a biologic competitor but rather as a central nervous system stimulant. The usefulness of methylphenidate lies in its ability to counteract the respiratory depression caused by a variety of anaesthetic and sedative agents, although other studies have shown it more effectively than this one.

Blood pressure changes were minimal in both groups of patients, and there was no opportunity to test the potentiating property in relation to epinephrine and norepinephrine as reported by Maxwell and associates.[8] Plummer and Yonkman[9] noted the same potentiation but in addition recorded that the hypertensive action of amphetamine and ephedrine was antagonized by methylphenidate.

The side-effects of emotional reactions were established in this series but were not so marked as in the reports of some other observers. Percheson[5] has described a case of almost maniacal reaction after the administration of 60 mg. intravenously. There may be some relation between dose, route of administration, and degree of psychomotor response, and since the methylphenidate was administered intramuscularly in the cases recorded in this paper, the sudden bombardment of intravenous therapy did not occur. Smith and Adrian[5] have listed nausea, disorientation, and garrulousness as some of the side-effects occurring after dosage of 0.2 mg./lb. body weight, but they were impressed by the analeptic effect of methylphenidate on barbiturate depression and/or poisoning.

Gale[2] considered one of the most gratifying effects of methylphenidate was its ability to stimulate depressed newborn infants. Out of eighteen infants, all but three responded within two minutes of administration of methylphenidate, 1 mg./5 lb. He also noticed that general body activity was increased, was at its maximum after 15 minutes, and maintained for about one hour. Therefore, the marked degree of irritation in the infant delivered by forceps mentioned in this study may have been owing partly to the difficult delivery and partly to the methylphenidate. However, in the three infants observed, the response to the methylphenidate was positive.

Summary

Methylphenidate (Ritalin®) has been studied in relation to its stimulant effect after anaesthesia. A control series was established by a double blind method of administration. A total of 294 patients were observed in the recovery room; 174 received methylphenidate and 120 normal saline. These patients had had only minor operations. On arrival in the recovery room, the blood pressure was recorded, a spirometer recording made, and then the patient received 1 c.c. of the unknown solution. The time of injection was noted carefully, and also the awakening time. A second recording was made with the spirometer 15 min. after the first one, whether the patient was awake or not. Analysis of the results indicated that there was no significant decrease in the awakening time of the methylphenidate series, but that the degree of respiratory stimulation due to the drug was statistically significant. Blood pressure changes were minimal and of no importance. Psychomotor stimulation occurred in both groups, but slightly more in the treated group than in the controls. Three depressed newborn infants responded to intramuscular injections of methylphenidate after the routine methods of resuscitation had failed to produce satisfactory results.

Résumé

Nous avons étudié le méthyphenidate (Ritalin®) et particulièrement ses effets stimulants après l'anesthésie. Nous avons fait l'étude avec une série de témoins en employant la méthode du double inconnu. Nous avons étudié un total de 294 malades dans la salle de réveil: 174 d'entre eux ont reçu du methylphenidate et 120 n'ont reçu que du sérum salé. Ces malades n'avaient subi que des opérations mineures. A l'arrivée à la salle de réveil, la pression artérielle était prise et notée, le volume des échanges était évalué au spiromètre et noté et, ensuite, nous donnions au malade 1 ml d'une solution inconnue. Nous notions le moment de l'injection soigneusement et ensuite le monent du réveil. Nous faisions également, 15 minutes après la première, que le malade fut réveillé ou non, une deuxième détermination du volume des échanges avec le spiromètre. L'analyse des résultats nous permet de conclure que, dans la série où le methylphenidate a été employé, le réveil n'a pas été plus précoce de façon marquée, mais nous pouvons affirmer que le médicament a produit une stimulation respiratoire importante. Les modifications de la tension artérielle ont été minimes et sans importance. Nous avons

observé une stimulation psychomotrice dans les deux groupes mais légèrement plus marquée chez les sujets traités. Trois nouveau-nés déprimés ont bien répondu à l'injection intramusculaire de methylphenidate alors que les méthodes ordinaires de resuscitation n'avaient pu donner de résultats satisfaisants.

ACKNOWLEDGMENTS

I would like to thank all the surgeons who very kindly gave their permission for their patients to be included in these trials. My gratitude is due to Miss Robins who made all the spirometer recordings and kept careful records, and to Mrs. Harms who gave all the medication in the recovery room in addition to making pertinent observations. The Chief Pharmacist and her staff played a vital part in making and issuing the solutions, so that our administrations would be random. Dr. Werner Kalow of the Department of Pharmacology of the University of Toronto once again very kindly advised me on the choice of statistical analysis. I am grateful to Ciba Company Limited for the supply of methylphenidate and for their generous contribution to the Research Fund. The Board of Governors of the Hospital have shown their usual interest and support in a research project.

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POTENTIATION OF THIOPENTAL ANAESTHESIA: WITH TIGAN®, PANECTYL®, BENADRYL®, GRAVOL®, MARZINE®, HISTADYL®, LIBRIUM®, AND HALOPERIDOL (R 1625)*

ALLEN B. DOBKIN, M.D.†

PREVIOUS STUDIES have shown that a therapeutic dose of most of the phenothiazines markedly prolongs thiopental anaesthesia, whereas a similar effect with narcotic analgesics is less pronounced.[1, 2] Two new anti-emetics, trimethobenzamide (Tigan®)[3] and haloperidol (R 1625),[4] and a new neurosedative, methaminodiazepoxide (Librium®),[5] were studied together with several commonly used antihistaminic drugs to determine also what effect they might have on thiopental narcosis.

METHOD

Crossover experiments were carried out four times at weekly intervals with each drug, using ten mongrel dogs of comparable age and size (13.2 kg. to 20.9 kg., mean 17.5 kg.). In every experiment each dog received 20 mg./kg. of thiopental intravenously in a 2 per cent solution, injected at the approximate rate of 150 mg. per min., and was followed at alternate experiments by the injection of the test drug. The dose selected for each drug was based on clinical experience or on information provided by the pharmaceutical manufacturer, and was adjusted so that severe cardiorespiratory depression would not occur when administered intravenously after thiopental.

RESULTS

Data from the experiments on each drug are summarized in Table I. The data from each series of cross-over experiments were analysed statistically to determine the probability of significance of the differences that were observed.

Trimethobenzamide (Tigan®) had no effect on thiopental narcosis. Thenylpyramine (Histadyl®) appeared to cause prolongation, but the effect was inconsistent, and the differences observed were not statistically significant. Dimenhydrinate (Gravol,® Dramamine®) caused only slight prolongation. Trimeprazine (Panectyl®), diphenhydramine (Benadryl®), cyclizine (Marzine®), and haloperidol (R 1625) caused moderate prolongation, whereas methaminodiazepoxide (Librium®) caused very marked prolongation.

No untoward effects were observed with any of these drugs except with haloperidol, which caused a disturbance of gait after recovery from anaesthesia. This effect was indicative of an extrapyramidal disturbance, which was observed also when this drug was administered to humans.

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TABLE I

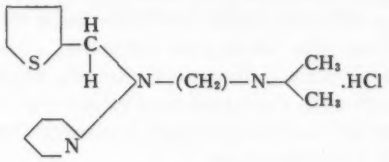
THE EFFECT OF SOME ANTI-EMETICS AND SEDATIVES ON THIOPENTAL NARCOSIS IN DOGS

	Dose mg./kg.	Recovery time (minutes) Head up Mean*	S.D.	Percentage diff.	Significance	Recovery time (minutes) Legs up Mean*	S.D.	Percentage diff.	Significance
Thiopental alone	20	55	23	−4	p = 0.5	73	32	−11	p = 0.4
+Trimethobenzamide	10	53	22			65	25		
Thiopental alone	20	44	11	+48	.001	53	9	+36	.001
+Trimeprazine	.25	65	15			72	14		
Thiopental alone	20	51	23	+63	.005	60	19	+62	.001
+Diphenhydramine	5	83	34			97	29		
Thiopental alone	20	39	12	+28	.025	47	9	+19	.005
+Dimenhydrinate	5	50	16			58	11		
Thiopental alone	20	35	9	+49	.001	43	9	+44	.001
+Cyclizine	5	52	12			62	12		
Thiopental alone	20	43	17	+25	0.1	51	19	+20	0.2
+Thenylpyramine	1	54	20			61	21		
Thiopental alone	20	47	18	+208	.001	56	17	+225	.001
+Methaminodiazepoxide	2.5	145	39			182	40		
Thiopental alone	20	42	13	+33	.001	54	15	+41	.005
+Haloperidol	0.5	63	21			76	25		

*Each mean time represents 20 administrations of thiopental alone and with the test drug.

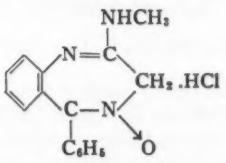
Discussion

According to these studies, the anti-emetic effect of trimethobenzamide will not be accompanied by any alteration in the hypnotic effect of thiopental, even if it is administered during anaesthesia.[3] On the other hand, the antihistaminic drugs all prolonged thiopental sleep.[4] If cyclizine is given during anaesthesia in order to prevent postoperative vomiting, it may reasonably be expected to prolong anaesthetic sleep considerably. If trimeprazine, diphenhydramine, or dimenhydrinate are administered parenterally during anaesthesia for a sedative or antihistaminic effect they may also significantly prolong sleep. Thenylpyramine HCL (Histadyl®), having the following structural formula,



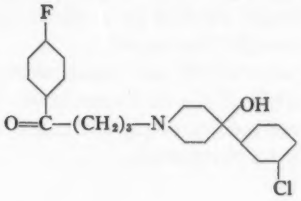
may be used parenterally during anaesthesia to treat acute urticaria due to drug allergy.[5] It would probably not prolong anaesthetic sleep if used in the dosage recommended.

Methaminodiazepoxide HCL (Librium®) has a structural formula as follows:



This compound was recently introduced mainly for the treatment of acute agitation and aggressive behaviour. The taming action of this drug is similar to chlorpromazine, and its muscle relaxing properties are greater than that of meprobamate. These effects occur apparently at a dose level which does not produce hypnosis. However, the present study indicates that when it is administered with thiopental, a very marked hypnotic effect is evident. This must be borne in mind when a patient who has been taking this drug must be given an anaesthetic.[6]

Haloperidol (R 1625) has the structural formula shown below.



This compound is one of a group of butyrophenones which have been under clinical trial as a psychosedative and an anti-emetic. Its psychic effect in man is not unlike chlorpromazine at a relatively low dose level which does not cause drowsiness. Unfortunately, it causes a high incidence of extrapyramidal nervous system disturbances which are very disturbing to the patient.[7] At the dose level tested, it causes moderate prolongation of thiopental sleep.

Summary and Conclusions

A standardized cross-over experiment was used on dogs with three recently introduced drugs—Tigan (anti-emetic), Librium (neurosedative), and Haloperidol (psychosedative and anti-emetic)—to determine whether they affect the duration of thiopental narcosis. These were compared with some antihistaminic and anti-emetic drugs (Panectyl, Benadryl, Gravol, Marzine, and Histadyl). The dose selected for each drug was based on clinical experience and was adjusted if necessary so that severe cardiorespiratory depression would not occur when they were administered after thiopental.

Tigan had no effect on thiopental narcosis. Histadyl caused only slight prolongation. Librium caused very marked prolongation. The other drugs caused slight to moderate prolongation of thiopental narcosis.

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Résumé

Nous avons employé, chez des chiens qui avaient absorbé récemment du Tigan (antiémétique), du Librium (neurosédatif) et de l'Haloperidol (sédatif psychique et antiémétique), une circulation croisée ordinaire pour étudier si ces médicaments modifient la durée de l'anesthésie au pentothal. Nous avons comparé les effets de ces médicaments avec ceux de certains antihistaminiques et antiémétiques (Panectyl, Benadryl, Gravol, Marzine et Histadyl). L'expérience clinique a servi de base pour déterminer la dose de ces médicaments et nous ajustions les doses de façon à éviter une dépression sérieuse du système cardiorespiratoire lorsqu'ils étaient injectés après une dose de thiopental.

Le Tigan n'a pas semblé avoir d'effet sur l'anesthésie au thiopental. L'Histadyl n'a prolongé que légèrement la durée de l'anesthésie. Le Librium l'a prolongée considérablement. Les autres médicaments ont causé une prolongation plus ou moins longue de l'anesthésie au thiopental.

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ANESTHÉSIE ET OPERATION CÉSARIENNE

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UNE QUESTION fort controversée selon les milieux, les habitudes, l'expérience et l'opinion personnelle des anesthésiologistes est celle de la technique idéale pour l'anesthésie au cours de l'opération césarienne. Ce qui importe, en somme, c'est d'administrer une anesthésie qui soit sans danger pour la mère et son enfant, tout en procurant au chirurgien une résolution musculaire suffisante pour qu'il puisse accomplir son travail dans les meilleures conditions possibles. Le but de la présente communication est d'établir la suériorité de l'anesthésie générale sur l'anesthésie rachidienne après une expérience portant sur 1,172 opérations césariennes pratiquées depuis neuf ans dans un hôpital spécialisé en obstétrique et gynécologie.

Pourquoi avons-nous graduellement, au cours de cette longue série d'interventions, abandonné presque complètement l'anesthésie rachidienne?

1. Si l'on considère la patiente, il faut se rappeler qu'il s'agit habituellement d'une jeune femme en bonne condition physique générale, mais plutôt assez nerveuse et fatiguée parce que son travail est déjà commencé depuis un laps de temps parfois assez considérable: sa nervosité sera augmentée par l'inquiétude provoquée chez elle quand elle réalise qu'elle ne peut accoucher normalement comme les patientes qui l'entourent, lorsqu'elle entend les chuchotements, murmures ou même conversations de son médecin avec le médecin consultant venu à son tour l'examiner; et qu'on l'amène au département de radiographie pour une pelvimétrie et enfin à la salle de chirurgie.

Pour toutes ces raisons, cette patiente acceptera moins facilement un mode d'anesthésie qui la gardera consciente durant une intervention tout de même assez dramatique, mais sera bien heureuse de se soumettre à une anesthésie générale qui, en quelques secondes, lui fera complètement oublier les circonstances pénibles où elle se trouve plongée.

2. Il ne faut jamais oublier la communication célèbre de Foster Kennedy[1] qui mettait les anesthésistes en garde contre la rachi-anesthésie lorsqu'il était possible de lui substituer l'anesthésie générale administrée de façon compétente: les complications neurologiques sont toujours redoutables, mais il me semble encore plus tragiques, si elles frappent une patiente aussi jeune que la plupart des femmes en couches.

3. C'est aussi l'opinion de Bergner, Roseman, Johnson et Smith[2] qui relèvent un nombre effarant de complications à la suite de l'anesthésie rachidienne, particulièrement lorsqu'elle est employée dans la spécialité de l'obstétrique.

4. Hingson et Hellman[3] recommandent une extrême prudence aux anesthésistes qui emploient la rachi-anesthésie en césarienne et déplorent un grand nombre de

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complications sérieuses à la suite d'injections de doses trop élevées qu'ils qualifient de «doses assassines»: Greenhill déclare que la rachi-anesthésie est la plus dangereuse anesthésie qui soit pour les femmes enceintes.[4]

5. L'anesthésie rachidienne au cours d'une opération césarienne, loin de protéger le fœtus contre le danger d'une hypoxie plus ou moins sérieuse, augmente au contraire ce danger (*a*) parce que l'action du diaphragme est diminuée par l'augmentation de volume de l'utérus gravide à terme, et (*b*) parce qu'elle provoque chez la mère une chute importante de la pression artérielle comme nous l'avons souvent constatée et cette opinion est corroborée par Forthman et Adriani[5] qui, sur 391 cas de césarienne sous rachi-anesthésie ont trouvé 82 per cent d'hypotension artérielle marquée, alors que sur 200 cas où l'anesthésie générale était administrée, ils n'ont jamais enregistré une seule chute de la pression artérielle.

6. Même si les complications graves ne sont pas trop fréquentes, il y a toujours le risque d'un céphalée plus ou moins persistante qui peut atteindre jusqu'à 15 per cent des patientes selon Geo. J. Andros et Harold D. Priddle[6] et il nous parait bien inutile d'ajouter ce désagrément à la suite de l'intervention grave qu'elles ont dû supporter.

Notre Technique d'anesthésie générale

Après avoir constaté au bout de quelques semaines d'observation que le temps maximum qui s'écoulait, entre le début de l'opération et l'extraction du nouveau-né ne dépassait jamais 4 à 5 minutes, nous avons adopté la ligne de conduite suivante:

La patiente reçoit comme pré-médication gr. 1/150 de sulphate d'atropine: elle est rassurée par nos explications et notre promesse de lui donner une anesthésie générale complète, sans danger pour elle ou pour son enfant, et qui lui assure un réveil rapide dès que tout sera terminé: durant ce temps le chirurgien procède au badigeonnage de l'abdomen, dispose ses champs opératoires, etc. et lorsqu'il est tout à fait prêt à commencer son intervention.

Nous injectons à la patiente 50 mg. de thiopental sodique et aussitôt après 30 mg. de succinylcholine et le chirurgien est avisé de procéder immédiatement pendant que nous administrons de l'oxygène pur au masque avec respiration manuellement assistée: d'une façon générale, ceci a été suffisant puisque dans moins de 4% des cas avons nous été obligés d'ajouter du cyclopropane dont l'administration n'est habituellement commencée qu'après la naissance du bébé et à ce moment, l'anesthésie générale est poussée comme pour une laparatomie usuelle.

En somme, le succès de cette technique, fort simple en soi, repose surtout sur la collaboration du chirurgien et de l'anesthésiste qui unissent leurs efforts pour arriver à un minutage parfait.

Nous procédions presque exclusivement de cette façon depuis 3 ans, quand nous tombe sous la main, en 1954, la 2è édition de Modern Practice in Anesthesia dans laquelle Frankis T. Evans[7] nous donnait son entière approbation en ces termes: «L'anesthésie générale par inhalations pour l'opération césarienne est le choix usuel et elle est supérieure à toute autre: l'habilité de l'anesthésiste est un facteur plus important que le choix de tel médicament, de tel appareil ou de telle technique: trop souvent melheureusement, les anesthésies administrées pour les

interventions chirurgicales en obstétrique, le sont par des résidents sans trop d'expérience ou d'habilité: il suffit d'une anesthésie très minime jusqu'à la naissance du bébé, après quoi l'anesthésie sera poussée davantage mais pas au point d'empêcher la contraction de l'utérus. »

L'anesthésie générale administrée de cette façon remplit bien les conditions exigées, à savoir: résolution musculaire adéquate, bonne contraction de l'utérus après l'extraction du foetus, aucune dépression respiratoire pour la mère ni pour le fœtus, absence complète d'effets toxiques, pas de chute de la pression artérielle.

Si, quelques uns craignent les vomissements et les complications pulmonaires qui en résultent au cours d'une anesthésie générale administrée à une patiente qui n'est pas toujours strictement à jeun, disons d'abord que nous n'en avons rencontrés que très rarement, puisque nous n'avons eu à pratiquer la succion que *22* fois sur un total de *1,172* cas de césarienne: et nous sommes persuadés que la raison de cette absence de vomissements soit le fait d'une anesthésie générale aussi peu profonde et aussi brêve: en effet, Bellville, Bross et Howland[8] ont étudié 3,794 dossiers et concluent qu'ils ont constaté infiniment moins de nausées et de vomissements chez les patients ayant reçu une anesthésie générale moins profonde et moins prolongée avec, au début de celle-ci, une injection de thiopental et d'une relaxant musculaire: ils ajoutent que la chute de la pression artérielle au cours d'une intervention augmente la fréquence et la gravité des vomissements.

Le rôle de l'anesthésiste ne se limite pas à administrer l'anesthésie à la patiente: il doit aussi assurer les soins adéquats requis par le bébé dès l'instant de sa naissance, à savoir: nettoyage complet des voies respiratoires supérieures, oxygénothérapie intensive suffisante si importante selon Eastman,[9] chaleur, etc.

Mais il faut ici insister sur un point d'une importance capitale: c'est la pratique de la succion de l'estomac du bébé pour éviter la régurgitation de liquides ou sécrétions dans les voies respiratoires au cours des heures qui suivent la naissance, avec les complications qui en résultent.

Gellis, White et Pfeffer[10] insistent sur l'importance de ce nettoyage complet de l'estomac du nouveau-né et rapportent une diminution considérable des complications pulmonaires rencontrées chez les enfants nés à la suite d'opération césarienne depuis qu'ils appliquent cette pratique de façon routinière.

D'une façon générale, les soins requis par le bébé ne sont pas très compliqués: il respire spontanément et réagit très favorablement parce que l'intervalle entre l'injection du thiopental et de succinylcholine et la naissance est trop court pour que ces médicaments puissent l'affecter comme l'ont prouvé d'ailleurs par leurs travaux Virginia Apgaret E. M. Pepper.[11]

Ceci suppose évidemment que le chirurgien opère avec une certaine rapidité: si les progrès de l'anesthésie n'exigent plus cette vitesse dans la majorité des interventions chirurgicales, il semble cependant que dans le cas d'une opération césarienne nous sommes justifiés de l'exiger et de pouvoir ainsi donner l'anesthésie générale plus agréable pour la mère et moins dangereuse pour elle et son enfant.

Les chiffres suivants nous permettent d'affirmer que l'ensemble de nos résultats opératoires justifie le choix de notre technique. De 1951 à 1960, 1,192 opérations césariennes furent pratiquées, dont 1,080 sous anesthésie générale: les indications

opératoires furent variables, la plus fréquente étant évidemment la dystocie pelvienne ou la disproportion céphalo-pelvienne, l'atonie utérine et le placenta praevia venant en deuxième place tout comme le remarque Edwin J. DeCosta[12] de North Western University de Chicago.

Le tableau suivant permet de constater que l'incidence du nombre des opérations césariennes par rapport au nombre des accouchements augmente considérablement avec les années et témoigne du progrès, de la valeur et de la qualité du service d'obstétrique.

TABLEAU I

Année	No. de césariennes	Mortalité maternelle	Mortalité fœtale	No. d'accouchements
1951	45	1	10	3,258
1952	59	1	10	3,958
1953	68	0	14	4,063
1954	122	3	20	3,623
1955	93	0	18	3,525
1956	125	1	9	3,789
1957	168	0	16	5,625
1958	181	2	19	5,757
1959	190	3	20	5,162
1960	121	2	11	3,102
TOTAL	1,172	13	147	41,862

Etudions maintenant les cas de décès, en commencant par ceux des mères: il faut remarquer que si 13 mortalités maternelles furent enregistrées, le facteur anesthésie, générale ou spinale, n'entre pas une seule fois en ligne de compte (Tableau II).

Il faut ici mentionner que nous sommes dans un hôpital où nous sont référés les mauvais cas rencontrés dans d'autres cliniques ou même dans quelques

TABLEAU II

Cas	Cause de mort	Combien de temps après l'opération	Indication opératoire
1	Mélanosarcome	5 jours	Mélanoblastome avec métastase généralisées—grossesse 6½ ms.
2	Syncope cardiaque	8 heures	Rupture utérine
3	Choc obstétrical	4 heures	Dystocie fœto-pelvienne
4	Embolie cérébrale	7 jours	Placenta praevia
5	Insuffisance cardiaque—maladie mitrale	6 jours	Dystocie du bassin—cardiopathie
6	Toxémie gravidique	5 heures	Dystocie du bassin—eclampsie
7	Collapsus cardio-vasculaire	8 heures	Dystocie de contraction
8	Hémorragie subite par inertie—choc irréversible	5 heures	Placenta praevia central total
9	Embolie pulmonaire	2 jours	Dystocie fœto-pelvienne
10	Lower Nephron Nephrosis	9 jours	Placenta praevia—hémorragie sévère
11	Oedème aigu pulmonaire—décompensation cardiaque	2 jours	Dystocie du bassin
12	Collapsus cardio-vasculaire Dégénérescence du myocarde	4 jours	Placenta praevia central total avec hémorragie abondante
13	Néphrite et hépatite toxiques aigues gravidiques—eclampsie	3 jours	Dystocie et éclampsie

maisons privées: il nous faut bien les accepter tels qu'ils nous sont présentés, même si parfois malheureusement les patientes sont pratiquement moribondes: en effet, sur ces 13 cas, 9 nous sont venus de l'extérieur et dans une condition si précaire que nous ne nous faisions aucune illusion sur le pronostic pratiquement fatal auquel nous devions faire face.

Parlons maintenant de la mortalité fœtale: sur 1,172 césariennes, nous constatons 147 cas de décès des bébés. De ces 147 décès, 59 étaient des bébés mort-nès; et pour 65 autres bébés dont le décès est survenu, entre quelques heures et quelques jours après la naissance, l'indication opératoire de la césarienne était toujours la même, à savoir, placenta praevia et l'âge de la grossesse variait généralement entre 6 et 7½ mois: ceci représente un total de 124 décès évidemment inévitables.

Ces constatations étant établies, il ne reste plus que 23 décès de bébés pour un total de 1,048 opérations césariennes, soit un pourcentage de 2.2 per cent, chiffre très satisfaisant et qui se compare avantageusement avec les chiffres cités par les meilleures cliniques canadiennes ou américaines.

Ces heureux résultats sont la preuve du succès que peut obtenir une bonne équipe chirurgicale travaillant en étroite collaboration avec les obstétriciens: nous sommes heureux ici de rendre à ces médecins et chirurgiens le magnifique témoignage que méritent leur compétence et le zèle au travail qu'ils ont toujours montrés.

Résumé

L'anesthésie générale est supérieure à tout autre mode d'anesthésie, quand il s'agit d'opération césarienne:

1. Elle est plus agréable pour la mère et sans danger pour elle ou son enfant.
2. Elle supprime le risque des complications neurologiques.
3. Elle ne provoque pas de chute de la pression artérielle.
4. Elle élimine le danger des vomissements post-opératoires.
5. Elle assure au chirurgien une anesthésie adéquate, un relâchement musculaire suffisant sans diminuer la qualité de la contraction utérine post-opératoire.
6. Elle exige cependant chez l'anesthésiste, habilité, expérience et collaboration entière avec le chirurgien.

Summary

The goal of this communication is to establish the superiority of general anaesthesia over spinal or any other type of anaesthesia for caesarean section, after a series of 1,172 cases extending over a period of nine years.

1. It is safer for both mother and child, and is more acceptable by the patient because of the psychic condition she is in when she has to undergo a caesarean section.
2. It does away with the risk of neurologic complications.
3. It does not produce any fall in the blood pressure nor any vomiting.
4. It gives good relaxation to the surgeon and does not interfere with uterine tonicity after operation.

5. It requires that the anaesthetist has the ability and experience and works with the entire collaboration of the surgeon to achieve perfect "timing."

The results, including both foetal and maternal mortality, are discussed and compare advantageously with published results in Canada and U.S.A.

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THE SIGNIFICANCE AND TREATMENT OF HYPERTENSION DURING ANAESTHESIA

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CIRCULATORY HOMEOSTASIS during anaesthesia is of great importance and every effort is made to maintain it. This is more commonly directed at avoiding extreme hypotension then hypertension, though the latter can be just as serious a phenomenon,[1, 10] a fact illustrated by the case reported here.

A weatherbeaten man of 62 years of age, 5 ft. 4 in. in height, and weighing 179 lb., was admitted for repair of a right inguinal hernia which had occurred four years previously during a tug-of-war. His exercise tolerance was still good in spite of many years' history of mild chronic bronchitis. On examination his chest was emphysematous in appearance, blood pressure taken on two occasions 145/90–196/90, E.C.G. normal, and chest X-ray normal.

At 10.30 A.M. morphine 10 mg. and atropine 0.4 mg. were given intramuscularly, and before induction at 11.45 he was awake and orientated with a systolic blood pressure of 160 mm. Hg and pulse of 88. Thiopentone 500 mg. was given slowly and anaesthesia continued with nitrous oxide, oxygen, and cyclopropane in a semiclosed circle absorption system. Succinylcholine 0.1 per cent was administered intravenously and artificial ventilation was used. During the ensuing 30 minutes the blood pressure rose to 280/120 and the pulse to 120. These cardiovascular signs preceded any other obvious evidence of ventilatory insufficiency or response to surgical stimuli. A diagnosis of phaeochromocytoma was dismissed at this time as unlikely and the patient was treated for hypercarbia, hypoxia due to emphysema, and mild bronchospasm. The soda lime was changed, patient intubated, artificial ventilation with a Jefferson ventilator (pressures -5 cm. H_2O—plus 20 cm. H_2O) begun, meperidine 25 mg. given, and the cyclopropane discontinued. Ventilation was now improved but severe bronchospasm gradually developed: after 15 min. the blood pressure had fallen to 220 mm. Hg: a supraventricular tachycardia of 130/min. and acute pulmonary oedema developed. This was treated by stopping the succinylcholine, of which 175 ml. had been used by this time, aspiration, reverse Trendelenburg tilt, cedilanid 1.6 mg. intravenously by intermittent injection, and voluntary respiration under ether anaesthesia. An hour later the pressure was 160/100, pulse still 150, and the operation completed. E.C.G. showed a nodal tachycardia and quinidine 0.6 gm. was given intramuscularly. The blood pressure gradually decreased to 80/70, but was maintained easily at approximately 115/70 with an L-noradrenaline drip.

On the following morning the patient was conscious, orientated, and ventilating well: his blood pressure was 140/80, but L-noradrenaline in small amounts was still necessary. E.C.G. was reported as a posterior wall myocardial infarct. During the day he became confused: the blood pressure was maintained, but death occurred forthy hours postoperatively.

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At post-mortem examination there was no macroscopic evidence of brain damage and no evidence of myocardial hypertrophy or recent infarction. There was bilateral pulmonary congestion and oedema with superimposed early bronchopneumonia of hypostatic type. The right adrenal gland contained a phaeochromocytoma. Death is considered due to a phaeochromocytoma causing hypertension, left ventricular failure, and pulmonary oedema, followed by hypovolaemia and adrenal failure.

The causes of a sustained hypertensive response during anaesthesia may be considered in two groups: firstly, those that can be anticipated and against which prophylactic measures taken and, secondly, those cases in which the response is anticipated merely as one of the possible complications of any anaesthetic. In the first group are patients suffering from phaeochromocytoma, thyrotoxicosis, transection of the spinal cord at or above T5, or a raised intracranial pressure. Also included are those who have received a vasopressor and to whom an oxytocic drug has subsequently been administered,[3] and those in whom the aorta has been clamped during the course of surgery.

The causes in the second group are more common and not infrequently act synergistically in the same case. Sensory stimuli during inadequate anaesthesia produce a pressor response even in the presence of hypovolaemia, though the blood volume must be within 20 per cent of the patient's predicted normal.[4] Clinical studies of cyclopropane anaesthesia have demonstrated a raised arterial pressure.[5, 6] This can occur with normal carbon dioxide levels and adequate depth of anaesthesia,[7] though in one series[5] hypercarbia was a constant finding. This in itself is associated with an increase in circulating catecholamines and a sustained elevation of pressure, the magnitude being unrelated to the increase in carbon dioxide tension.[8, 9] In the early stages of pulmonary oedema there are increases in pulse rate and arterial pressure, and the skin is usually cold and sweaty.[2]

There are now at least twenty-seven reported cases in which patients undergoing surgery for a variety of reasons were found to have had an unsuspected phaeochromocytoma.[10–18] The anaesthesia was usually complicated by hypertension and pulmonary oedema. Twenty-three patients died within 48 hours postoperatively, frequently much sooner, and the diagnosis was made or confirmed at post-mortem examination.

The hazards of a sustained hypertension are that a cerebrovascular accident, cardiac failure, cardiac arrhythmia, or pulmonary oedema may occur. Also, it has been demonstrated in the experimental animal that raising the blood pressure can reflexly increase the bronchoconstrictor tone.[19] The pressor response is sometimes associated with a tachycardia which in itself is harmful. The ventricular filling time in diastole is reduced, lowering the cardiac output. The minute volume of any mitral regurgitation is increased and the competent right ventricle augments the volume of blood in the lungs, increasing the possibility of pulmonary oedema. Patients suffering from arteriosclerosis, mitral valvular disease, or pulmonary hypertension are especially prone to these ill effects. The toxic effects of adrenaline have been reviewed by Millar,[20] and Szakacs[21] and King[22] have produced myocardial damage experimentally in dogs by the intravenous administration of L-noradrenaline in clinical dosage.

Treatment of a causative process is preferable to symptomatic treatment; and if the hypertension is considered with reference to the rate of development, peripheral circulation, ventilation, pulse rate, and other signs of autonomic activity, the cause may readily be determined. However, the degree and duration of hypertension that can be tolerated by a patient cannot be predicted with certainty, and possibly a sustained pressor effect for any reason during surgery plays a significant role in postoperative morbidity. In view of the hazards mentioned, it is advisable to take active steps to reduce a hypertension, when it occurs, even though the underlying cause cannot at the time be definitely decided. It is noteworthy that of the 27 reported cases of unsuspected phaeochromocytoma, three survivors[11, 13, 14] were those for whom therapeutic measures had been directed primarily at correcting the pressor response and if these were unsuccessful the operation was abandoned.

Various drugs have been advocated for the prophylaxis or treatment of hypertension in anaesthesia under different circumstances: chlorpromazine,[3, 23, 24] hexamethonium,[1, 25, 26] trimethaphan,[14] and phentolamine.[10, 13, 14] The following plan of campaign is suggested when, for reasons not fully understood, the systolic blood pressure rises at least 40 mm. Hg above that normal for the patient, and maintains that level. If successful it should enable the operation to be completed safely and the diagnosis of any additional pathology made during the convalescent period.

1. Ensure adequate ventilation but avoid a phase of negative pressure in the respiratory cycle at this time.

2. Reduce the rate of administration of intravenous fluids unless the patient is known to be hypovolemic with a healthy myocardium.

3. If there are confirmatory signs of inadequate anaesthesia, increase the concentration of the anaesthetic agent in use. In the absence of such signs, administer chlorpromazine 5–10 mg. intravenously and determine the effect of postural changes on the blood pressure.

4. If the blood pressure persists at the original high level, administer phentolamine 2–5 mg. intravenously and continue by intravenous infusion if necessary.[27, 28] This drug is used routinely for diagnostic purposes and for the treatment of hypertensive crises during the surgical removal of a phaeochromocytoma, but should be administered with caution. It has been postulated that a decreased red cell mass and chronic hypovolaemic state may be present in patients with phaeochromocytoma, and investigation of two patients substantiate this. Consequently, additional therapy required may be restoration of the patient's blood volume.[29]

5. Supportive cardiac therapy with digitalis or prostigmine[30, 31] may now be considered. Only when the pressor response has been brought under control should consideration be given to using a negative pressure phase in artificial ventilation.

Summary

A case report of a 62-year-old man undergoing general anaesthesia for an inguinal herniorrhaphy is presented. Hypertension, bronchospasm, tachycardia, and pulmonary oedema developed during the procedure and the patient was

treated for left ventricular failure. Death occured 40 hours postoperatively and a phaeochromocytoma on the right side was discovered at post-mortem examination. An account of the causes and significance of hypertension during anaesthesia is given; and a plan of treatment suggested for these cases in which the reason for the pressor response is difficult to determine. This involves the assessment of ventilation and depth of anaesthesia, use of chlorpromazine and phentolamine. Attention is drawn to the possible need for additional cardiovascular supportive therapy.

RÉSUMÉ

Nous avons présenté le cas d'un homme de 62 ans soumis à une anesthésie générale pour une opération de cure de hernie inguinale. Au cours de l'opération, il a présenté de l'hypertension, un bronchospasme, de la tachycardie et de l'œdème pulmonaire, puis ce malade a été traité pour insuffisance ventriculaire gauche. Il est mort quarante heures après l'opération et l'on a trouvé, à l'autopsie, un phéochromocytome droit. Nous avons émis des opinions sur les causes et la signification de l'hypertension au cours de l'anesthésie; nous suggérons une ligne de conduite pour traiter ces cas où il est difficile de préciser la raison de cette manifestation d'hypertension. Cela doit inclure une évaluation de la ventilation et de la profondeur de l'anesthésie et l'usage de la chlorpromazine et de la phentolamine. Nous attirons également l'attention sur la nécessité éventuelle d'appliquer une thérapeutique additionnelle pour supporter le système cardiovasculaire.

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HALOTHANE-AIR ANAESTHESIA USING THE "PULMOTEC" APPARATUS

PRELIMINARY REPORT

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DUE TO the extreme potency of halothane the usual range of clinical concentration is very low (0.5–2.5 per cent). This suggests that vaporization with air should be possible without materially compromising the oxygen supply to the tissues, since the gas displaced by the halothane would be mostly nitrogen. A glance at the oxygen-haemoglobin dissociation curve shows that minor variations in PO_2 at the right-hand side between 80–100 mm. Hg have only minor effects upon the percentage saturation of haemoglobin.

In the event of mass casualties from atomic warfare and so on, it is quite possible that in many areas the supply of compressed gases would fail. As far as anaesthesia is concerned, this has important implications when one realizes that without compressed gases an anaesthetic machine is as useless for anaesthesia and resuscitation as an automobile without gasoline. While the loss of gases would still leave open drop, local, regional, and intravenous techniques, when controlled or assisted ventilation is mandatory some means of inflating the lungs could be life saving.

The recent introduction of the thick-walled foam rubber bag and automatic non-rebreathing valve, in such examples as the Ruben Resuscitator from Denmark using the Ruben valve and the Pulmonator Resuscitator from the United States using the Lewis-Leigh valve, suggests a ready application to the problem. Either of these devices in conjunction with halothane in a Fluotec vaporizer should provide us with a simple portable draw-over type of anaesthetic machine, completely independent of compressed gases, having a controllable anaesthetic concentration and no explosion hazard.

DESCRIPTION OF APPARATUS

Figure 1 is a photograph of the assembled apparatus. It consists of the following:

1. A Pulmonator Resuscitator (Western Anaesthesia Equipment Co.), made up of a rubber bag inside which is a thick-walled foam plastic liner. The capacity of the bag is 5 litres (2 L. size is also available). At either end of the bag is a short length of hollow plastic tube which projects some distance into the interior. On the back end is a unidirectional flap valve moving air always in a forward direction. The Lewis-Leigh valve fits on the front end of the bag and is designed

*Assistant Clinical Professor of Anaesthesiology, University of B.C. Department of Anaesthesia, Vancouver General Hospital, Vancouver, B.C.

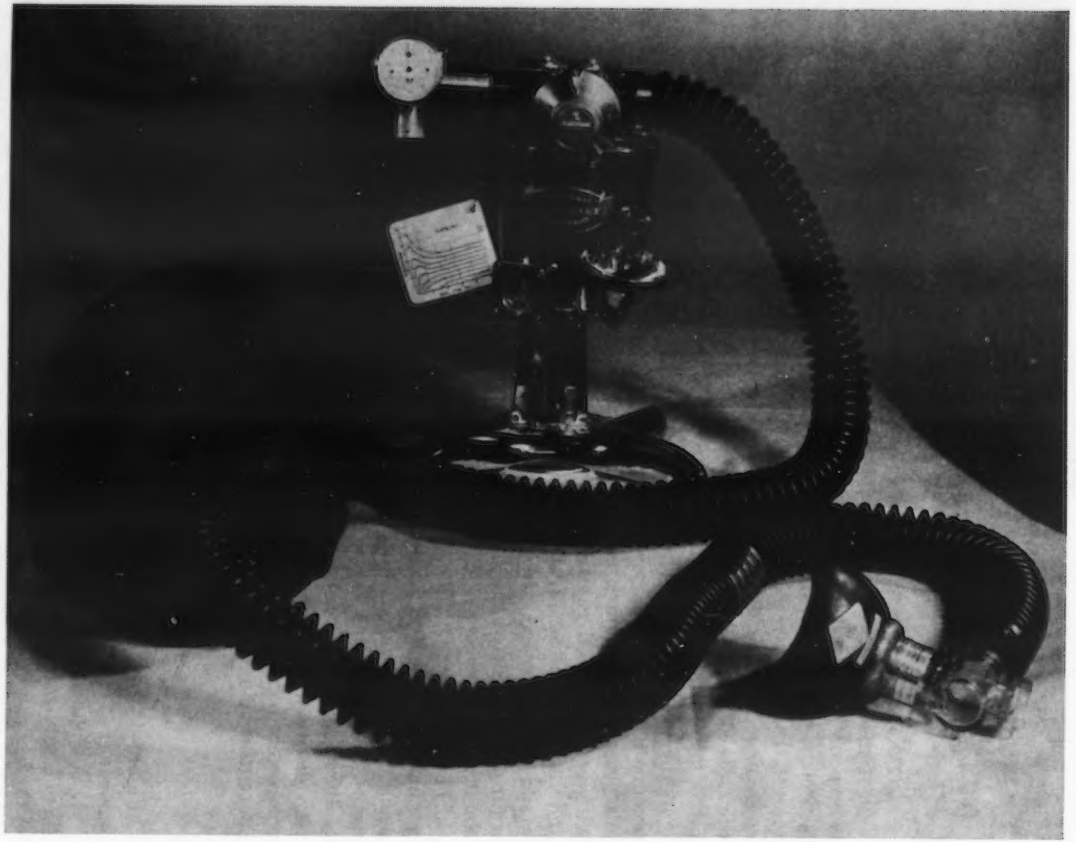

FIGURE 1

in such a way that compression of the bag forces a siliconized rubber flap to occlude the exhalation tube, the end of which is cut on an angle. Thus air can only move outwards through the mask. During exhalation, the rubber flap falls back against the inhalation opening and expired air can leave only through the exhalation tube. The author has modified the valve by adding an additional flap on the outside of the exhalation vent to ensure that with spontaneous breathing air can be drawn in only via the vaporizer. Both the M.I.E. and Heidbrink female adaptors will fit the valve, as well as a 15-mm. male endotracheal connector.

2. A Mk. II Fluotec halothane vaporizer on a stand with suction cup base.

3. Two lengths of standard anaesthetic hose. It is clear that the bag may be placed at any point behind the valve, and it was found convenient to have it remote from the face.

4. A Wright anemometer was added to the apparatus and placed at the air intake side to avoid moisture laden exhaled air and halothane vapour passing through it.

Selection of Cases

The series consists essentially of 102 consecutive list cases. Ages ranged from 18 months to 89 years. The types of operation performed, which were mostly of a major nature, are shown below.

Classification of 102 Cases

E.E.N.T.	20	Major Plastic	5
Major Abdominal	19	Varicose veins	5
Urology	13	Radical mastectomy	2
Major Orthopedics	8	Radical neck dissection	2
Gynaecology	6	Thoracotomy	1
Neurosurgery	6	Thyroidectomy	1
Dental	6	Cardiac catheterization	1
Breast Biopsy	6	Amputation of legs	1

Technique

Premedication was as follows. Adults received pentobarbital 1½ to 3 gr. two hours preoperatively; morphine ⅛ to ¼ gr., and atropine 1/150 to 1/50 gr. one hour preoperatively. Children and the aged were premedicated in accordance with weight and general condition.

Induction and Maintenance

The majority of cases received thiopentone intravenously in standard dosages. Where intubation was performed succinylcholine 20–60 mg. was used.

Blood pressure was measured in the usual way with a cuff on the upper arm, and was monitored very frequently along with pulse rate, tidal and minute volumes.

Early in the series it was clear that the most satisfactory technique was to hyperventilate patients with 2 to 3 per cent halothane following induction.

FIGURE 1

Gradually the concentration was reduced until after 10 to 20 minutes a maintenance setting of 0.5 to 1.5 per cent was quite satisfactory.

With light premedication higher maintenance concentrations were generally needed, and conversely with heavy sedation.

Where a lower setting than 2 per cent was used immediately following induction, patients frequently reacted to the surgery, requiring either additional thiopentone, meperidine, or a rapid increase in concentration.

In two cases, halothane-air was employed for induction but was slow and unsatisfactory. In children, however, halothane-air induction was often very rapid and in one case the child was easily intubated after two minutes of 2 per cent halothane. As with halothane generally, blood pressure always fell to a lower level, but the hypotension was never severe enough to require a vasopresser. Hypotension could be avoided by intravenous atropine 1/100 gr. during induction and remedied when required either by atropine or by lowering the concentration unless reaction to surgery was thereby provoked.

Usually, a fall in blood pressure to around 80 to 90 mm. Hg was well tolerated and produced a dry surgical field. Spontaneous ventilation could be readily overridden and controlled, and, in fact, it was very simple to wash out enough CO_2 to produce apnoea for a considerable period.

Abdominal relaxation was often adequate with hyperventilation and halothane, but when unsatisfactory was achieved by intermittent intravenous succinylcholine. In a few instances intermittent gallamine was used.

Minute volumes of 8 to 10 L. could be maintained with the greatest of ease and tidal volumes of up to 2,000 c.c. were readily produced by squeezing the pulmonator bag. Once a steady maintenance concentration setting was achieved, spontaneous ventilation was generally quite adequate as judged by tidal and minute volume readings in relation to age and weight. Blood colour was quite acceptable. Where ventilation was below the predicted normal value it was assisted manually.

It was noted that beyond the 2.5 per cent concentration setting on the Fluotec, resistance to flow increased appreciably. However, this presented no problem as at this setting, or greater, spontaneous ventilation required assistance anyway, and with controlled ventilation the only effect was a slowing in the rate of refilling of the bag. Since maintenance was generally at a much lower setting the fact was of little practical importance.

Blood Colour

Although estimation of arterial O_2 saturation by the visual method is far from quantitative, so long as ventilation was adequate in accordance with the Radford nomogram arterial blood appeared satisfactorily pink. Generally, patients were hyperventilated to be on the safe side. However, when ventilation was stopped or allowed to be deficient, cyanosis rapidly became obvious. With air vaporization it can probably be assumed that in the steady state if the blood appears pink the P_{CO_2} level is within the normal range. This is in contrast to hypoventilation with high oxygen percentage mixtures where high oxygen saturation and high P_{CO_2} levels can be present simultaneously.

Halothane Consumption

This was estimated by measuring the exact amount of halothane needed to bring the vaporizer liquid level back to its original mark after using the same setting for periods of one hour and over. Using controlled ventilation 8 to 10 L./min. the following consumptions were found: 1 per cent: 10–15 c.c./hr.; 1.5 per cent: 15–20 c.c./hr.; 2.0–2.5 per cent: 20–25 c.c./hr.

There were no untoward events in the series, and the incidence of postoperative nausea and vomiting was very low. Three patients complained of nausea and two of nausea and vomiting which could be attributed to the anaesthetic. There was one death in the series. This was a 75-year-old woman who was admitted moribund with severe toxaemia following a ruptured appendix. She died 24 hours following laparotomy, but it is unlikely that anaesthesia played any part in her death.

Special Cases

Two men with severe emphysema were anaesthetized for cystoscopy with this technique and were allowed to breathe spontaneously throughout with intermittent assistance. There was no tendency to apnoea as with high oxygen concentrations and both were awake and talking on completion of the surgery.

An 18-month-old child was anaesthetized for cardiac catheterization by this method and it proved most satisfactory, with no interference with blood gas studies. She had previously been investigated under local anaesthesia and arterial oxygen saturation was 93 per cent. An identical figure was obtained during halothane-air anaesthesia with spontaneous respiration.

Conclusion

The "Pulmotec" is a simple portable apparatus capable of delivering a very satisfactory anaesthetic without the need for compressed gases. Apart from any use it might possibly have in emergency situations, it has proved very satisfactory for anaesthesia in dental offices where portability and rapid nausea-free emergence is invaluable. Since vaporization takes place only during the inhalation phase, halothane consumption is quite moderate. An additional advantage is that the apparatus without the vaporizer is a very effective resuscitator. Since there is no rebreathing, soda-lime is unnecessary. The manufacturers of the Fluotec (Cyprane Ltd., England) have assured me that even at flow rates of 30 L. per min. through the vaporizer, concentrations still remain at the indicated setting. The apparatus can be further simplified by using the smaller vaporizers such as the Rowbotham or Goldman, though as accurate a calibration would not be possible as with the "Fluotec."

In a further study it is planned to follow arterial blood oxygen saturation levels during halothane-air anaesthesia.

Summary

A simple portable apparatus has been described for the administration of halothane with air vaporization.

The equipment consists of a Pulmonator Resuscitator, a Mk. II Fluotec vaporizer, and a Wright ventilometer. While designed mainly for possible military field use and national catastrophe, it will provide a very satisfactory non-explosive anaesthetic without the need for compressed gases.

Possible indications for its peacetime use in hospitals would be severe emphysema, to avoid apnoea from high oxygen concentrations, and cardiac catheterization where the use of N_2O interferes with blood gas analysis.

In the interests of economy or improved portability, simpler vaporizers can be successfully employed. The Pulmotec is also instantly available as a resuscitator where anaesthesia is not required.

Acknowledgment

The author wishes to acknowledge the assistance rendered by Ayerst, McKenna and Harrison, Montreal, who provided the equipment for this study.

Résumé

Nous avons décrit, ci-contre, un appareil portatif pour l'administration de l'halothane vaporisé avec de l'air.

L'installation consiste en un pulmonateur resuscitateur, un vaporisateur Fluotec Mark II et un ventilomètre Wright. Bien que cet appareil soit construit particulièrement pour usage possible en campagne militaire et en cas de catastrophe nationale, il peut permettre l'administration très satisfaisante d'une anesthésie sans risque d'explosion et sans recourir à des gaz sous pression.

Les indications possibles de son usage en temps de paix dans nos hôpitaux seraient: les cas d'emphsème marqué, la crainte de produire l'apnée en employant de grandes concentrations d'oxygène dans l'atmosphère que le malade respire et, au cours des cathétérismes cardiaques, l'élimination du protoxide d'azote qui modifie les résultats des analyses de gaz dans le sang.

Des vaporisateurs plus simples peuvent être employés avec succès; il y en a de plus économiques et plus facilement portatifs. S'il n'est pas nécessaire d'administrer de l'anesthésie, le pulmotec est utilisable instantanément comme resuscitateur.

LETTERS TO THE EDITOR

University Hospital,
University of Saskatchewan,
Saskatoon, Saskatchewan,
January 16, 1961.

SIR:

I have read with great interest Dr. Michael Johnstone's paper on the azeotrope (CASJ 8(1):53 [Jan., 1961]). I have the highest respect for Dr. Johnstone and his impressive experience with halothane. Since we have administered at the University Hospital in Saskatoon well over 4,000 anaesthetics with the azeotrope and have done some experimental work as well, I feel, however, that I may be justified in taking issue with some of his remarks.

1. I believe it has been proven in the pages in this Journal (7(3): 297 [July, 1960]) that in clinical ranges the azeotrope vapour is non-flammable in both oxygen and air. If Dr. Johnstone has had the experience of a fire with this agent I can only assume that either the mixture used was not the azeotrope but contained an excess of diethyl ether or else that the concentration exceeded the limit of non-flammability. As for ignition of the liquid by static electricity this must be a small hazard indeed in a well-equipped and disciplined operating suite.

2. It has never been claimed that hypotension does not occur with the azeotrope. However, from our experimental work, it would appear quite clear that hypotension with the azeotrope is a sign of deep anaesthesia whereas equally profound hypotension can be produced with halothane in the presence of comparatively light planes of anaesthesia (CASJ 7(2):91 [April, 1960]). The peripheral stasis which Dr. Johnstone has observed is probably due to just that fact, namely, deep anaesthesia. We have managed to induce controlled hypotension of a moderate degree for prolonged periods of time with halothane/ether without circulatory ill-effects, where such hypotension was desirable from the surgical point of view.

With halothane, overdosage is relatively easily produced and avoidance requires the purchase of special vaporizers, whereas the contrary is true for the azeotrope as long as it is used with reasonable caution; this confers upon the azeotrope a safety factor not inherent with halothane. This greater safety factor matters little in Great Britain where the overwhelming majority of anaesthetics are administered by or under the supervision of highly skilled consultants, but it is of the very greatest importance in all those countries (and that includes Canada and the United States) where so much of the anaesthetic work is done by untrained or semi-trained personnel and where, if halothane were used widely by these people, a substantial increase in anaesthetic mortality might well result. It is the duty of teachers and leaders of the specialty to point the way to safer anaesthesia for all patients in all hands and not overly emphasize those agents and techniques which only the skilled can master with safety.

3. Lastly, I do not believe that Dr. Johnstone's experimental evidence is based on sound reasoning. I should have been more convinced if in an equal number of cases he had reversed the sequence of agents, preceding the administration of halothane by the azeotrope. I am not quite sure how much reliance one can place on data obtained from an experiment such as the one reported, since it is well known that all anaesthetics exert a certain amount of effect in subclinical doses and therefore it is proper to assume that at the time the azeotrope was administered, at least traces of halothane were still circulating in the patient and exerting what may well have been a synergistic effect.

GORDON M. WYANT, F.F.A.R.C.S.

State University of New York
Upstate Medical Center
Syracuse 10, N.Y.

SIR:

As one of the participants in the early clinical and laboratory studies of both halothane and the halothane-ether azeotrope, I believe that the current article by Dr. Johnstone, Evans, and Murphy (CASJ, Jan. 1961) deserves some comment, as it might leave the reader with some very gross misconceptions.

First, the authors do not seem to have a clear idea of what an azeotropic mixture is. Halothane and diethyl ether form an azeotropic mixture only when particular proportions are used (68.3 to 31.7 V/V halothane to ether) and is not "azeotropic in all proportions." The minor conflagration experienced by M.J. indicates either that he did not have *the azeotropic mixture* but probably one with excessive ether, excessive halothane, or else he was using an exceedingly high concentration—which should be necessary only rarely. If his mixture had excessive ether, he was probably administering a pure ether anaesthetic *in high concentration.*

Second, the entire design of his comparison of halothane-oxygen and the azeotrope-oxygen leaves so much to be desired that it is ludicrous to comment on the results he describes.

After working for several years with those who are not skilled in the administion of anaesthetics, a teacher may gain insight into which agents are easier (in a physiological-pharmacological sense) to administer. In my association with Professor Wyant, we were both impressed by the tendency of our trainees to prefer the *azeotropic mixture* to the use of halothane, but perhaps such consideration has no more merit than making a pronouncement after using any new anaesthetic agent twenty-one times *after having reached a deep anaesthetic state with another very potent agent* (viz., *halothane*)!

There are many ways of administering an anaesthetic, and the use of halothane-oxygen is only one of these. In very skilled hands, the outcome is usually satisfactory to the administrator as well as to the patient. In witnessing and partaking

in many hundreds of administrations of *the azeotropic mixture* as well as of halothane and trying to use the common sense expected of a trained anaesthetist, I believe that the use of the halothane-ether azeotrope is less illogical in clinical practice than the use of halothane, even though a skilled anaethetist can provide excellent anaesthesia with either of these.

ALLEN B. DOBKIN, M.D.

NEWS LETTER

British Columbia Division

Dr. Frank McCaffrey has been appointed Director and Head of the Department of Anaesthesia, St. Paul's Hospital, Vancouver, B.C. Dr. McCaffrey replaces Dr. C. Roach who has retired.

The new 500-bed Lions Gate Hospital in North Vancouver opened this spring with an anaesthetic staff of six Certified anaesthetists. Dr. R. Townsley was appointed Head of the Department at that hospital.

The Second Annual Refresher Course for General Practitioners conducted by the Department of Anaesthesia of the University of British Columbia and its affiliated hospitals was held on January 25–27, 1961. Forty general practitioners from British Columbia, Alberta, Washington and Oregon registered. The Guest Speaker was Dr. H. B. Fairley of the University of Toronto and the course was an outstanding success.

The British Columbia Division continues to maintain an active scientific programme throughout the year. The following meetings have been held:

August, 1960, Guest Speaker, Mr. Forrest Bird, Bird Respirator Company.

September, 1960, Dr. D. J. Pearce, Department of Anaesthesia, Southampton General Hospital, Southampton, England, on "Respiratory Insufficiency."

October, 1960, Dr. E. Daniels, Department of Pharmacology, University of British Columbia on "Recent Advances in Pharmacology of Interest to the Anaesthetist."

November, 1960, Dr. David Power, St. Mary's Hospital, Montreal, Que., on "A New Technique for the Control of Labor Pains."

January, 1961, Dr. H. B. Fairley, University of Toronto, on "Respiratory Problems."

February, 1961, Dr. J. Abajian, Professor of Anaesthesia, University of Vermont, on "Fluothane Studies."

Dr. J. V. Taylor who has completed his Fellowship training at the University of British Columbia has joined the staff of the Department of Anaesthesia at the University of Washington, Seattle.

Dr. W. A. Doll, formerly on the staff of the Edmonton General Hospital has joined Associated Anaesthetic Services and the Department of Anaesthesia at the Vancouver General Hospital.

Dr. Guy Screech who recently completed his training at the University of British Columbia has joined Associated Anaesthetic Services and the Department of Anaesthesia at the Vancouver General Hospital.

Dr. F. G. Williams who recently completed his training at the University of British Columbia has joined the staff of the new Lions Gate Hospital, North Vancouver.

Dr. M. Hiddleston, formerly on the staff of St. Mary's Hospital, New Westminster has joined the staff of the new Lions Gate Hospital, North Vancouver.

Dr. Irene Millar who recently completed her Fellowship training at the University of British Columbia has joined the staff of the new Lions Gate Hospital, North Vancouver.

Dr. J. B. Harland, formerly on the staff of the University of Saskatoon Hospital, has established private practice in Anaesthesia in Kelowna, B.C.

Saskatchewan Division

The Advisory Planning Committee of the Province of Saskatchewan on Medical Care, the "Thompson" Committee, which has been charged by the provincial government to submit its recommendations for a comprehensive medical care programme in Saskatchewan, has recently concluded public hearings in Regina and Saskatoon. Amongst others, briefs were presented by the College of Physicians and Surgeons of Saskatchewan and by the College of Medicine of the University of Saskatchewan. The brief by the College of Physicians and Surgeons contained a sub-brief submitted by the Section on Anaesthesia of the College, which outlines in detail the history of the speciality of anaesthesia in this province and sets forth the views of the members of the Section on future developments. The brief of the College of Medicine which dealt with problems of medical education and research was a consolidated submission in which the views of the Department of Anaesthesia were included.

Dr. T. Pimblett having completed his training at the General Hospital, St. John's, Newfoundland, has been appointed Instructor in Anaesthesia at the University Hospital, Saskatoon, with effect from January 1, 1961.

Manitoba Division

The Winipeg anaesthetists were hosts during the month of February to Dr. M. Holmdahl, Professor of Anaesthesia at the University of Uppsala, Sweden.

Dr. John Abajian, Professor of Anaesthesia, University of Vermont, visited the Manitoba Division during the month of February.

Dr. T. Johanasson has joined the Department of Anaesthesia at the Misericordia Hospital in Winnipeg.

Ontario Division

The University of Toronto has announced the appointment of Dr. R. A. Gordon as Head of the Department of Anaesthesia in the rank of Professor, to succeed Dr. S. M. Campbell on his retirement on July 1, 1961.

Dr. D. W. S. Best has been appointed Chief of Service in Anaesthesia and a member of the Board of Governors of the Joseph Brant Memorial Hospital, Burlington, Ontario. This 240-bed hospital opened its doors on February 1, 1961. Associated with Dr. Best are Dr. J. K. H. Culverhouse, formerly of Cornwall,

Ontario, and Dr. J. E. Marshall and Dr. D. C. Aikenhead, both of Burlington.

The Department of Anaesthesia of Queen's University have included the following guest speakers in their 1960–61 programme: Dr. E. H. Botterell, Dr. H. B. Fairley, Dr. R. W. Gunton, and Dr. S. M. Campbell of Toronto; Dr. A. B. Noble, Montreal; Dr. D. M. Little, Hartford, Connecticut; Dr. J. P. Payne, London, England.

The Ontario Division will hold a meeting at Kingston on October 6 and 7, 1961. This meeting will be preceded by a three-day refresher course for general practitioner anaesthetists, to be presented by the Department of Anaesthesia of Queen's University.

Quebec Division

About 50 anaesthetists attended the winter meeting of the Quebec Division, which was held on February 18, 1961, in the amphitheatre of the Montreal General Hospital. The guest speaker, Professor E. Trier Mörch, gave a delightfully entertaining lecture on "Hyperventilation." He stressed the importance of ventilation, not only during anaesthesia, but also as an integral part of the treatment of ventilatory insufficiency owing to such conditions as crushing chest injuries, bulbar polio, and barbiturate poisoning. Professor Mörch enlivened his presentation not only by his own inimitable wit, but also by showing a moving picture depicting the effect of ventilatory pressures on the micro-circulation in the wing of the bat. Dr. John Price, of the resident staff of the Montreal General Hospital, also delivered a paper concerning anaesthesia in upper abdominal surgery. Business meetings of the Executive Committee and of the Division as a whole were also held, as well as a reception and dinner for the members and their wives. There were no speeches.

Division du Québec

Samedi, le 18 février 1961, avait lieu au Montreal General Hospital, la réunion du printemps de la section du Québec de la Société Canadienne des Anesthésistes.

(*a*) A la réunion d'affairs tenue l'avant-midi, le président sortant de charge, le docteur David Power, annonçait que la division du Québec avait enfin obtenu depuis quelques mois son incorporation. Le docteur Léon Longtin était élu à l'unanimité le nouveau président de la section du Québec—Sincères félicitations et tout le succès désiré.

Les docteurs Roger Gagnon et J. B. I. Sutherland du Comité Economique permanent faisaient rapport de leurs entrevues récentes avec les différentes compagnies d'assurance, avec l'Association Médicale Canadienne, avec l'Association des Médecins de Langue française. Une liste des honoraires minima, basés sur l'opération pratiquée a été envoyée au Collège des Médecins et Chirurgiens de la province de Québec; elle doit être étudiés par cet organisme en présence des représentants de la spécialité incessamment. Une fois acceptés, cette liste d'honoraires servira de base aux pourparlers futurs avec les différents organismes avec lesquels nous aurons à traiter.

(*b*) Dans l'après-midi, avait lieu la réunion scientifique. L'invité d'honneur était le docteur Trier Mörch, de Chicago University, qui a évidemment traité de ventilation. Le docteur Mörch, avec le dynamisme qu'on lui connait, a parlé de l'utilité du ventilateur dans les traumatismes thoraciques avec fractures multiples; il a également démontré l'effet de la ventilation sur la circulation. Son exposé était illustré de deux films des plus démonstratifs.

(*c*) La réunion a été clôturée par un souper succulent, dans une atmosphère de cordialité, où il fait plaisir se retrouver aussi souvent que l'occasion s'en présente.

Les 6 et 9 février, nous êtions les invités à l'Hôpital saint-Joseph de Rosemont de la Société de Physiologie et de Pneumatologie de Montréal. Les docteurs André Cournand, de l'Université Columbia, Pierre Nadeau et François Laramée de l'Hôtel-Dieu de Montréal. A. E. DuBois, D. V. Bates et Maurice MacGregor du Royal Victoria étaient les conférenciers invités. Les sujets traités ont été du plus grand intérêt pour nous anesthésiologues: tableau clinique des insuffisances respiratoires; ventilation et perfusion pulmonaires dans l'emphysème; diagnostic et traitement de l'insuffisance respiratoire aigue; troubles circulatoires dans la défaillance respiratoire.

Les 13, 14 et 15 février derniers, le docteur Louis Lamoureux organisait pour la région de Montréal, à l'Hôpital Notre-Dame, un symposium sur le Fluothane. Les membres du Service d'Anesthésie de l'Hôpital Notre-Dame, les docteurs Guy Fortin, Kieri-Szanto, Gilles Cossette, François Lafleur, R. Denis, Jean-Paul Brunelle, Jean-Paul Lavallée et Jean-Luc Perreault prenaient part tant aux travaux théoriques qu'aux démonstrations cliniques. Les docteurs John Abajian, professeur d'anesthésie de l'Université de Vermont, et le docteur G. B. Gilbert, professeur d'anesthésie de l'Université McGill, étaient les conférenciers invités.

Les docteurs Léon Longtin, Eug. Allard, et Simon Dombrowski ont fait dernièrement un stage d'étude à Washington pour assister aux cours du Dr. S. N. Albert, sur la détermination des volumes sanguins au moyen de l'Isotope Cr 51.

Le Dr Jean Guy Patoine, après avoir passé avec succès ses examens au Collège Royal du Canada et au Collège des Médecins et Chirurgiens de la Province de Québec, fait maintenant partie de l'équipe de Service d'Anesthésie de l'Hopital de l'Enfant-Jésus à Québec.

BOOK REVIEWS

CUTANEOUS INNERVATION. Edited by WILLIAM MONTAGNE. New York: Pergamon Press. 1960. $10.00.

THIS VOLUME contains the Proceedings of the Brown University Symposium on Biology of Skin, 1959. The twelve contributors have produced a series of most informative and stimulating reviews of current knowledge of cutaneous innervation, ranging through the minutiae of anatomical, physiological, and pharmocological concepts of the subject. This symposium should be of value to both the clinician and research worker as an authoritative review of contemporary knowledge in this field.

R.A.G.

PRINCIPLES OF GENERAL NEUROPHYSIOLOGY RELATING TO ANAESTHESIA AND SURGERY. By B. D. WYKE. Toronto: Butterworth & Co. Ltd. 1960. $4.25.

DR. WYKE'S detailed contribution to *General Anaesthesia* by Evans and Gray has now been published as a separate entity. The author points out that he has aimed at selectivity rather than over-all coverage. However, his readers will agree that those aspects of neurophysiology which he has selected are most ably covered. The material is collected under the headings Cellular Neurophysiology, Interneuronal Communication, and Cerebral Circulation and Metabolism. The reviewer felt that considerably more value could have been gained from the many references by including names in the text, rather than as an unassociated list at the end of chapters. The price of the book is minimized by making no changes from the major text. Thus, there is no index and no reference to the myoneural junction, the latter being discussed by another contributor in the major text.

H.B.F.

HYPOTHERMIA AND THE EFFECTS OF COLD. British Medical Bulletin, January 1961.

SEVEN CANADIANS have contributed to this issue of the British Medical Bulletin, which provides much interesting and detailed information on the latest aspects of Hypothermia. It suffers from the lack of continuity of any multiple author publication but has the advantage, in many instances, of contributions from international authorities.

The fourteen sections range in coverage from the Resistance of Poikilothermic Animals to Cold to the Sensitivity of Hypothermic Animals to X Irradiation. Between these extremes there are, among others, sections on Hibernation, Clinical Techniques, and Clinical and Experimental Profound Hypothermia and contributions on the physiology of the various systems under conditions of hypothermia. The authors give a total of 757 references, although many of

these are repeated from section to section. This symposium is to be recommended to all those interested in this subject.

ANAESTHETIC ACCIDENTS. By V. KEATING, M.B., B.CH., D.A., F.F.A.R.C.S. Chicago: Year Book Publishers Inc. (Toronto: Burns and MacEachern Ltd.). 1961. $5.50.

THE SUB-TITLE, "The Complications of General and Regional Anaesthesia," is more descriptive of the contents of this 280-page book than the title "Accidents." Written for those not in close touch with a medical library, the chapters give a great deal of physiological and pharmacological information, particularly about the circulatory and respiratory systems, which provides a background for the treatment of the complications mentioned. The author's advice regarding prophylaxis, diagnosis, and treatment is practical and useful. One of his suggestions for safety is warning bells on all gas cylinders to indicate emptiness.

This is not a book to be opened in the operating room when in difficulties, but it should aid an anaesthetist in becoming familiar with signs which indicate danger, either before, during, or after anaesthesia.

S.A.F.

MEETINGS

CANADIAN ANAESTHETIST'S SOCIETY, ANNUAL GENERAL MEETING
Seigniory Club, Montebello, P.Q.
May, 15–18, 1961

CANADIAN MEDICAL ASSOCIATION, NINETY-FOURTH ANNUAL MEETING
Queen Elizabeth Hotel, Montreal, P.Q.
June 19–23, 1961

ONTARIO DIVISION
CANADIAN ANAESTHETISTS' SOCIETY
Kingston, Ontario
October 6–7, 1961

MEMBERSHIP LIST, 1961

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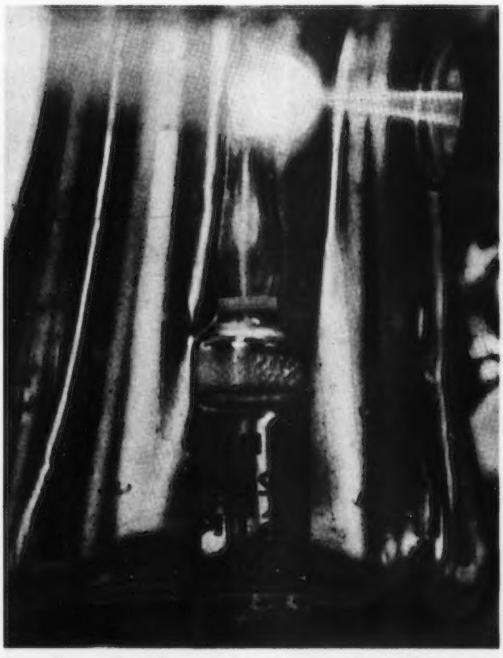

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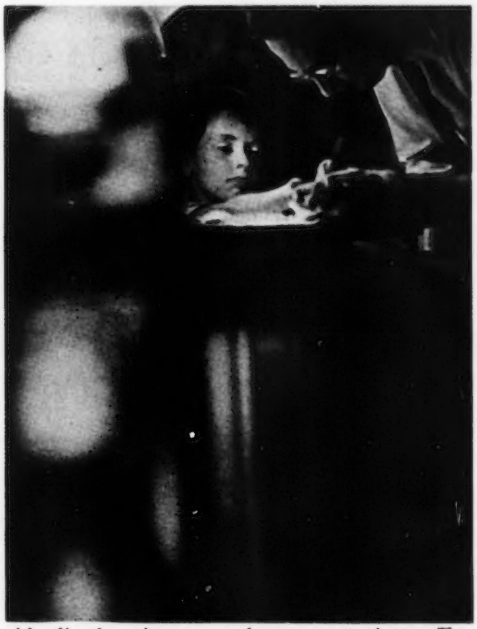

vidualized volumes and concentrations. For general use Xylocaine is recommended in concentrations of 0.5%, 1% and 2%, with the 2% solution generally used for nerve block. Minimal volumes of 4% Xylocaine may be used topically in those cases where lower concentrations are ineffective or inadequate. The 4% solution (in ampoules of 5 cc.) may also be used transtracheally and for retrobulbar injection.

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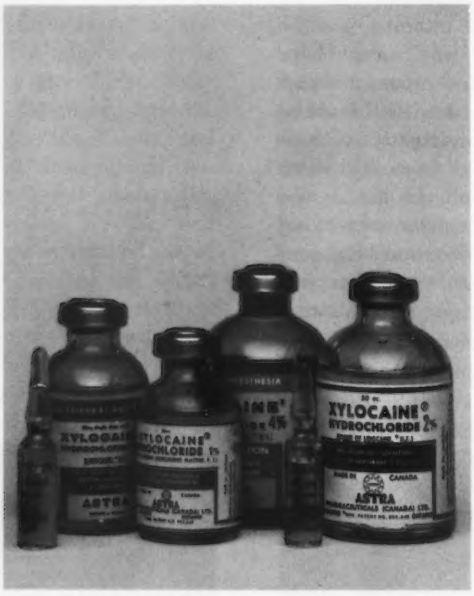

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‡ Bryce-Smith, R.: Local analgesic drugs. Brit. M.J. 1:1039 (April 2)1960.

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